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## *ABSTRACT*

Title of Thesis: Psychological and Metabolic Correlates of Obesity in African-Americans and Caucasians

Christie S. Oates, Master of Science, 2006

Thesis directed by: Martha M. Faraday, Ph.D.

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The purpose of the present study was to identify whether there are unique biological, behavioral, psychological, and environmental factors specific to African-Americans that may promote the development of obesity. Chronic stress levels and the hormonal and metabolic responses of 63 Caucasian and African-American men and women to two metabolically-relevant events — a metabolic load (standardized meal) and a metabolic demand (standardized exercise) were assessed. The hormonal and metabolic responses included hypothalamic-pituitary-adrenal axis hormones (i.e., adrenocorticotropin hormone and cortisol) and insulin responses to a meal. African-Americans reported higher levels of perceived chronic stress, but had lower plasma levels of the stress hormone cortisol than did Caucasians at baseline and throughout both testing sessions. Acute insulin responses and total insulin production to a meal were significantly higher among African-Americans compared to Caucasians. Striking ethnic differences emerged in the psychological factors that mediate responses to stress and predict health behaviors, such that African-Americans reported less social support, less rest/sleep, and more negative appraisal than Caucasians. Overall, the biological (i.e., acute insulin responses and total insulin production in response to a meal) and psychological findings (i.e., higher chronic stress, less social support, less rest/sleep, and

more negative appraisal) in overweight but otherwise healthy African-Americans compared to healthy, overweight Caucasians suggest a high vulnerability for the early onset of metabolic disorders such as obesity.

# Psychological and Metabolic Correlates of Obesity in African-Americans and Caucasians

by

Christie S. Oates

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To my dear significant other, our story is just beginning.

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# INTRODUCTION

Obesity results from a complex interaction of biological, psychological, social, environmental, and cultural variables (Pi-Sunyer, 2002). Genetics may contribute to approximately 30%-40% of an individual's body mass index (BMI). Non-biological variables contribute about 60-70% to an individual's BMI (Pi-Sunyer, 2002). The influences of these various factors may be difficult to separate. For example, obesity is more prevalent among women and minority groups, with African-American and Hispanic women having the highest rates (CDC, 2006). These different prevalence rates may reflect genetic differences, such as genetically-determined metabolic differences (Greenberg, Dintiman, & Oakes, 1998). In addition, the differences may reflect sociocultural differences, such as exposure to stress, social support, health habits, and cultural ideals for health and beauty. Ethnic differences in obesity prevalence also may be the consequence of biological and environmental factors.

Little is known about the causes of ethnic differences in obesity prevalence. In fact, the majority of obesity literature has focused on Caucasian-Americans. The high prevalence rates of overweight and obesity among African-Americans and the relative ineffectiveness of available treatments indicate the need to better understand the etiology of energy imbalance across ethnic groups.

This master's research project focused on factors that might contribute to ethnic differences in obesity prevalence. The material below reviews the relevant literature with regard to African-American versus Caucasian-American differences in obesity prevalence, the rationale for examining specific metabolic responses and environmental variables, and how ethnic differences in chronic stress levels may affect energy balance,

and, subsequently, overweight and obesity. The impetus for this research project is the high prevalence of obesity in African-Americans and the goal of identifying whether there are unique biological, behavioral, and environmental factors specific to this subgroup that may promote the development of obesity.

## Obesity

*Definitions.* Overweight is an excess of body weight derived from muscle, bone, fat, and/or body water (NIDDK, 2005). Obesity is defined as excess of body fat (Anderson & Wadden, 1999). Overweight and obesity are most often estimated by calculating an individual's BMI (NIDDK, 2005). BMI of 25-29.9 $kg/m^2$ is considered overweight and over 30 $kg/m^2$ is obese (NIDDK, 2005). Obesity has been stratified into three classes (class 1, BMI 30-34.9 $kg/m^2$; class 2, BMI 35-39.9 $kg/m^2$; class 3, BMI $\geq$ 40 $kg/m^2$) (NHLBI, 1998). The term *obesity* is used throughout this paper to include all three classes.

*Impact of Obesity.* In 1999, the Centers for Disease Control and Prevention warned that obesity "should be taken as seriously as any infectious disease epidemic" (Hall & Jones, 2002, p. 657). Over the last several decades, Americans have shifted toward high fat diets, modern energy-saving conveniences, and increasingly sedentary lifestyles. All of these factors contribute to the obesity epidemic. In fact, the incidence of obesity has increased by one-third in the past decade (Friedman, 2003). Further, approximately 65% of adult Americans are either overweight or obese (Flegel, Carroll, Ogden & Johnson, 2002) and 60% of adults do not engage in regular physical activity (USDHHS, 1996). In fact among adults, 32.2% are obese and 4.8% are considered

extremely obese (Ogden et al., 2006). The prevalence of overweight among children and adolescents has doubled and tripled, respectively, in the last two decades (Stein & Colditz, 2004). The problem of excess weight is not confined to the U.S. Across the globe, obesity rates have reached epidemic proportions such that the World Health Organization declared obesity as "one of the greatest neglected public health problems of our time" (Björntorp, 1997; Hall & Jones, p. 657, 2002).

*Prevalence in African-Americans and Caucasians.* The prevalence of overweight in the United States is about 35% for both African-Americans and Caucasians, 41% for Hispanics, and 26% for Asians (CDC, 2001). However, obesity disproportionately affects African-Americans. Approximately 30% of Caucasian adults are obese compared to 45% of African-American adults (CDC, 2006). The striking difference in the obesity prevalence rates between these two ethnic groups is present as early as 12-19 years old. In this particular age cohort, African-American male and female youth are 21% more likely to be overweight compared to 14% of Caucasian American male and female youth (CDC, 2004). This alarming trend among African-American youth is a harbinger of poor health outcomes associated with obesity.

*Health and Economic Burden.* Obesity poses major health risks, and is the seventh leading cause of preventable diseases. It is implicated in over 112,000 deaths each year in the U.S. (Flegal, Graubard, Williamson, & Gail, 2005; Mark, 2005). Obesity is often a precursor for medical problems (i.e., hypertension, insulin resistance, hyperlipidemia) or a cluster of these problems, known as metabolic syndrome (Pijl, 2003). Consequently, the economic burden of obesity as a result of hospitalization, lost worker productivity, and premature death is estimated at $117 billon annually (Stein &

Colditz, 2004). Despite a $30 billion industry, the most widely-used treatments for overweight and obesity in the U.S. are largely ineffective (Anderson & Wadden, 1999; Wadden, Brownell, & Foster, 2002). Approximately 75%-80% of formerly obese individuals fail to maintain weight loss (Byrne, 2002), and most dieters return to their pretreatment weight and gain additional weight (Garner & Wooley, 1991; Perri, 1998). Weight cycling disrupts homeostasis and may lead to maladaptive metabolic responses, such as a 15-20% lower metabolic rate, increased vulnerability to develop cardiovascular disease, and greater likelihood to gain more body fat (Garner & Wooley, 1991). The pattern of weight cycling may negatively affect the habitual dieter's overall health such that experts recommend maintaining current body weight rather than striving for weight loss (Garner & Wooley, 1991; NHLBI, 1998).

**Biological Factors**

This section reviews the relevant biological factors that contribute to obesity. More specifically, the roles of HPA axis hormones (e.g., ACTH and cortisol) and insulin in the development of obesity are reviewed. Together, these hormones regulate energy and maintain blood glucose levels. Furthermore, the extant literature suggests that African-Americans and Caucasians differ in their metabolic responses to daily events such as rest, feeding, and physical activity (e.g., Palaniappan, Carnethon, & Fortmann, 2002; Yanovski et al., 1996; 2000).

*Metabolic Responses. The hypothalamic-pituitary-adrenal (HPA) axis.*

Cortisol, one of the primary regulators of the HPA axis, is an important energy-regulating hormone. The HPA axis consists of the hypothalamus, the pituitary gland, and adrenal gland. These target organs communicate via hormonal signals, such that the

hypothalamus releases corticotropin-releasing factor (CRF), which signals the pituitary to make adrenocorticotropin hormone (ACTH). In turn, ACTH stimulates the adrenal cortices to make and release cortisol (Marieb, 1998). Rising levels of cortisol serve as a negative feedback signal to the hypothalamus, which reduces CRF production. A decrease in the release of CRF, in turn, reduces pituitary release of ACTH, which decreases adrenocortical output of cortisol. The secretion of cortisol has a distinct episodic and diurnal pattern that occurs throughout a 24-hour period (Weitzman et al., 1971), with peak levels attained subsequent to rising in the morning and lowest levels in the evening hours preceding and just after sleep (Weitzman et al., 1971; Marieb, 1998).

Although best-known as a stress hormone, cortisol serves critical energy regulating functions in the basal, non-stressed state. Cortisol, a glucocorticoid, is named for its ability to increase the level of glucose in the bloodstream. Cortisol regulates the body's energy stores through the processes of gluconeogenesis and lipolysis (Epel, Lapidus, McEwen, & Brownell, 2001). Glucocorticoids are essential to ensure relatively stable levels of circulating glucose during normal cycles of feeding, resting, and activity – events that vary in terms of energy demand and expenditure. Cortisol is also critical in the stressed state to maintain energy availability and allow the individual to resist and cope with physical, metabolic, and psychological stressors (Marieb, 1998). In the presence of an appropriately functioning HPA axis, cortisol responses are tightly regulated and quickly diminish with cessation of or adaptation to a stressful experience (Guyton & Hall, 2000).

*The HPA axis and body weight regulation.* During the last decade, there has been increased focused that obesity involves dysregulation of the HPA axis. The literature on

the HPA axis and obesity is complicated and somewhat contradictory. Contradictions may be explained, to some extent, by methodological differences among studies, such as regard for diurnal variations in cortisol secretion and whether the HPA axis was stimulated by inducing stress or by specific hormonal agents (i.e., dexamethasone, corticotrophin-releasing hormone, or hydrocortisone), or perhaps by differences in subject populations (including differences in age, gender, ethnicity). In particular, detection of differences between obese and normal weight individuals in HPA axis function appears to depend on whether the axis was assessed basally, in a stimulated state, and/or across the diurnal cycle. Numerous studies report basal cortisol levels in obese individuals that are less than normal weight controls (Jessop, Dallman, Fleming, & Lightman, 2001; Korbonits et al., 1996). Elevated basal cortisol levels also have been reported in obese individuals (Pasquali & Vicennati, 2000; Stunkard, Faith, & Allison, 2003). The relationship between elevated cortisol and weight is strongest in individuals with abdominal obesity (Chrousos, 2000).

*Diurnal variation.* There also is evidence to suggest that the cortisol diurnal rhythm is abnormal in obese individuals, with levels higher than normal during the night and lower than normal at rising from sleep (Björntorp & Rosmond, 2000; Duclos et al., 2001; Jessop et al., 2001). Other studies note normal diurnal cortisol patterns in obese individuals but differential HPA reactivity when challenged with pharmacologic agents. Jessop and colleagues (2001) administered saline, 7.5 mg of hydrocortisone, or 15 mg of hydrocortisone over a 24-hour period to obese Caucasian males and normal weight Caucasian males. At baseline, obese subjects had greater mean ACTH concentrations but lower mean cortisol concentrations than normal weight controls. After hydrocortisone

was administered, normal weight subjects had appropriate HPA axis suppression with significantly lower ACTH levels during the day and during the night. Obese subjects demonstrated suppression of ACTH only during the day but not at night. The HPA axis of obese individuals, therefore, was insensitive to feedback control during the night – a finding that could be related to the increased propensity for fat deposition among obese individuals. If cortisol levels are higher than normal during the night, then it may be that dysregulation of HPA axis activity in obese individuals is associated specifically with the distribution of adipose tissue and/or abnormal diurnal variations in cortisol.

*Response to a meal.* Differences have been detected between normal weight and obese individuals when stimulating the HPA axis by ingesting a meal. Korbonits and colleagues (1996) found hypocortisolemia in obese individuals at baseline but significantly greater cortisol levels after a meal and longer recovery times to pre-meal baseline levels for obese subjects compared to lean subjects. In addition, women with abdominal obesity had greater elevations in ACTH and cortisol after a meal challenge compared to their counterparts with peripheral obesity (Korbonits et al., 1996).

*Hypercortisolemia common among obese and depressed individuals.* The interpretation of cortisol levels is complicated by the finding that a subgroup of obese individuals may be hypercortisolemic, in part, because they also suffer from psychological disorders (Chrousos, 2000). In particular, depressive illness has been consistently associated with hypercortisolemia (Stunkard et al., 2003). Approximately 25%-30% of obese patients are depressed or have symptoms associated with mental illness (Anderson & Wadden, 1999). There is evidence to suggest that the relationship between abdominal obesity, mood disorders, and carbohydrate craving is mediated by

deficits in serotonin and elevations in cortisol (Chrousos, 2000; Björntrop & Rosmond, 2000). Administering antidepressants increases the number of glucocorticoid and mineralcorticoid receptors, which increases sensitivity of the negative feedback loop and lowers concentrations of ACTH and cortisol. The implication is that serotonergic drugs can modify a dysregulated HPA axis, thereby decreasing abdominal obesity and improving depressed mood (Björntrop & Rosmond, 2000). Whether obese individuals respond differently from non-obese individuals to antidepressants is unclear. In the existing literature, BMI of patients treated with antidepressants is generally not reported. Given that the HPA axis also regulates the response to stress, and that stress has been implicated in the precipitation and exacerbation of psychological disorders, HPA axis dysregulation may explain part of the etiology and comorbidity of psychopathology and obesity (Holsboer, 2000).

*The HPA axis and chronic stress.* Approximately 40% of poor health in adults can be attributed to chronic stress (Phillips, Kiernan, & King, 2001). Chronic overactivation of the HPA axis has been implicated in the initiation and exacerbation of physical, as well as psychological illness, including abdominal obesity, clinical depression, and diabetes (e.g., Stunkard et al., 2003). Over time, the effects of dysregulated stress responses and, in particular, elevated cortisol levels are evident in the brain and periphery. Such a pattern also affects immune, cardiovascular, and metabolic systems (McEwen, 2004). In particular, maladaptive responses to stress may overload homeostatic systems and subsequently change hormonal set points and other controls that maintain an organism's steady state (McEwen & Wingfield, 2003). Several studies suggest that the hippocampus, which is integral to recover from stress by inhibiting the

HPA axis, begins to atrophy. Atrophy of the hippocampus has been associated with recurrent depression and Cushing's Syndrome — disease states that share a common feature of hypersecretion of cortisol (McEwen, 1998). With regard to obesity, stress-induced cortisol secretion is linked with increased appetite and preferences for foods high in calories (Epel et al., 2001). Other investigators have found that high urinary cortisol levels were associated with high levels of dietary restraint (McLean, Barr, & Prior, 2001). Taken further, some individuals may decrease food consumption under stressful conditions (Grunberg & Straub, 1992).

It is important to note that a pattern of hyperreactive physiologic responses to stressful conditions occurs in depressed as well as in obese individuals. Short-term physiologic response of obese individuals after a metabolic stressor (e.g., a meal) is greater activation of the HPA axis than in normal weight individuals. If this increased response persists for long periods of time, then it is possible that HPA axis dysregulation is important in the etiology of obesity. It may be that depression and obesity are actually different phenotypic expressions of a common dysregulated neurobiological process.

*Ethnic differences in HPA axis responses.* Relatively few studies have compared HPA axis responses of African-Americans and Caucasians. The few existing studies have revealed large differences in physiologic and endocrine responses in naturalistic and laboratory settings. For example, one naturalistic study examined the cortisol response of overweight African-Americans and overweight Caucasians to awakening (Bennett, Merritt, & Wolin, 2004). Adult men and women provided two morning saliva samples separated by 30 minutes. Despite similar BMI, African-Americans had significantly lower levels of cortisol at awakening than Caucasians. Interestingly, *post hoc* analyses

revealed that cortisol levels were significantly lower among African-Americans with minimal education compared with more highly educated African-Americans. After adjusting for perceived stress, higher educated Caucasians had substantially higher cortisol levels than any group (i.e., Caucasians with low education, African-Americans with low or high education) at the 30-minute sampling. In this case, the more highly educated Caucasians demonstrated exaggerated HPA activity. These results are inconsistent with the extant literature for two reasons: (1) it is generally accepted that cortisol secretion is inversely proportional to socioeconomic status (e.g., Kapuku, Treiber, & Davis, 2002; Ockenfels et al., 1995); (2) the consensus is that African-Americans report greater levels of perceived stress and poorer health outcomes (e.g., Williams, 1999). However, the implication of this study is that African-Americans have a blunted HPA response that is likely to be exacerbated by low socioeconomic status when compared to Caucasians.

The laboratory studies conducted by Yanovski and colleagues, in which participants' HPA axis was stimulated via hormone administration or exercise, are the most frequently cited (Yanovski, Yanovski, Harrington, Gold, & Chrousos, 1995; Yanovski et al., 2000). Prior to hormonal stimulation, Caucasian and African-American women had comparable ACTH levels. However, after an exogenous dose of CRH, African-American women had higher levels of ACTH, whereas levels of cortisol (plasma and free) and cortisol-binding globulin were similar to Caucasian women (Yanovski, Yanovski, Gold, & Chrousos, 1993; Yanovski et al., 1996). Similar findings have been reported in African-American men versus Caucasian men by the same investigators. In response to exogenous CRH, African-American men exhibited markedly elevated ACTH

levels from 30-180 minutes post-administration compared to Caucasian men (Yanovski et al., 1995). Despite large differences in ACTH levels, total plasma cortisol concentrations were similar.

Taken together, these findings suggest that during and after stressful conditions, African-Americans are likely to exhibit an HPA response characterized by ACTH levels higher than those of Caucasians with cortisol levels similar to those of Caucasians. The similarity of HPA axis responses between African-Americans and depressives is interesting. Under basal conditions, depressives have a blunted ACTH response, but when stimulated with CRH, their response is exaggerated (Holsboer, 2000). In contrast to African-Americans, depressives also hypersecrete cortisol. It is important to note, however, that studies to date on HPA axis differences between African-Americans and Caucasians have assessed only non-depressed, normal weight African-Americans. Because obesity is associated with hypercortisol secretion, it is possible that obese African-Americans also hypersecrete cortisol. This possibility has yet to be examined.

***Insulin.*** Insulin is a hormone produced by the pancreas. Its primary function is to lower the amount of glucose in the bloodstream by facilitating the uptake of glucose into the cells. Several tissues throughout the body have receptors that respond to insulin including skeletal and cardiac muscle, liver, and mammary glands (Pike & Brown, 1986). Individuals with a fewer number of insulin receptors, receptor sites that are less responsive to insulin, or receptor sites that are less effective at the uptake of insulin have high circulating levels of glucose in the bloodstream. These conditions characterize insulin resistance – wherein insulin produced by the pancreas is largely ineffective in lowering blood glucose levels. Hyperinsulinemia is often a compensatory strategy for

insulin resistance. If excess glucose remains in the bloodstream once energy demands are satisfied, then insulin promotes storage of the excess energy as fat (Guyton & Hall, 2000).

*Differences in levels of insulin by body type and ethnicity*. Concentrations of insulin and glucose are higher in obese individuals compared to normal weight controls (Pasquali et al., 1999), both when assessed after fasting and when assessed in response to an insulin or glucose infusion. These metabolic differences between obese and normal individuals were demonstrated within 30 minutes of stimulating insulin and glucose responses.

There are similarities in insulin responses to a metabolic demand among obese individuals and non-obese African-Americans. Epidemiologic studies have demonstrated that fasting insulin levels were significantly higher in African-Americans compared to Caucasians at normal and overweight BMI categories (Palaniappan, Carnethon, & Fortmann, 2002). This relationship was especially pronounced in African-American women. Similarly, Melby and colleagues (2000) reported that normal-weight African-American women exhibited greater insulin resistance in response to an intravenous glucose tolerance test compared to Caucasian women of similar body types. However, the two groups did not differ in fasting insulin or glucose levels. Taken together, these findings suggest that metabolic responses associated with energy metabolism may be different among obese people and non-obese African-American individuals compared to Caucasians.

***Summary of similarities between non-obese African-Americans and obese individuals.*** There are interesting parallels in biological factors (especially diurnal

variation of cortisol levels and insulin responses to a meal) among African-Americans and obese individuals. Obese individuals are hypocortisolemic in the morning and African-Americans have lower morning cortisol than Caucasians (Bennett et al., 2004). Whether African-Americans also have other HPA axis responses that are similar to obese individuals (such as cortisol levels that peak at night and that are higher and prolonged in response to a meal) is unknown.

African-Americans and Caucasians have different ACTH levels in response to exercise and pharmacologic stimulation (e.g., in response to a dose of CRH), with African-Americans having higher ACTH responses but similar cortisol levels. Contrary to the negative feedback loop mechanism that characterizes hormonal responses among Caucasians, rising ACTH levels in response to exercise is not associated with the subsequent rise in cortisol among African-Americans. Because it is not clear whether these differences in HPA axis responses among African-Americans also promote obesity, this research project compared the relationship between BMI and HPA axis responses in African-Americans and Caucasians. Because hypercortisol secretion is associated with depression, the present study excluded participants with a history of psychopathology and used the Beck Depression Inventory (BDI-I; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961a) to assess the presence of current depressed mood symptoms among included participants.

Non-obese African-Americans, like obese individuals, have higher fasting insulin levels compared to non-obese Caucasians (Palaniappan et al., 2002). Further, African-Americans require more insulin to remove the same amount of glucose from the bloodstream than did Caucasians (Haffner et al., 1996). At lower body weights (i.e.,

normal and overweight), African-Americans produce higher insulin levels in the fasted state and when stimulated by a metabolic load compared to Caucasians. Because hyperinsulinemia is associated with obesity, this research project matched African-American and Caucasian participants by weight category and compared insulin responses to a standardized meal.

**Environmental Factors**

One environmental factor that may partially explain the ethnic differences in the prevalence of obesity is chronic stress, resulting from racial and ethnic discrimination, perceived racial and ethnic discrimination, or low socioeconomic status. Numerous studies suggest that African-Americans report higher levels of chronic stress compared to Caucasians (Fiscella & Franks, 1997). This may reflect perceived or actual racial and ethnic discrimination.

*Ethnic differences in levels of chronic stress.* African-Americans report high levels of chronic stress from factors that disproportionately affect them, including unemployment, low socioeconomic status, limited health care access, and racial discrimination (Fiscella & Franks, 1997). In 2003, the official poverty rate in the U.S. across all racial and ethnic groups was 12.3 % (U.S. Department of Commerce, 2004). The poverty rate among Caucasians is 8.2% and 24.4% among African-Americans. In a 2001 Gallup poll, 47% of African-Americans reported unequal treatment while engaged in their daily activities such as shopping and dining at restaurants. However in this same survey, 69% of Caucasians responded that there was no difference between the treatment of Caucasians and African-Americans. In contrast, 59% of African-Americans perceived themselves as treated worse than Caucasians. More than half of the African-Americans

surveyed perceived unequal housing and labor force conditions as ethnic discrimination (Clark, Anderson, Clark, & Williams, 1999). It is this perception of unequal treatment among African-Americans that Brondolo (2006) – described as a "background stressor." In other words, the perception of racial discrimination held by many African-Americans can be considered a chronic stressor that looms in the background, co-existing with other stressors (e.g., family, occupational) common to all ethnic groups negative effects on psychological and physical well-being (George & Lynch, 2003).

**Behavioral Factors**

*Racism-specific coping responses.* Brondolo and colleagues (2005) also reported that increased exposure to racism is associated with an increased tendency to interpret future events as racially discriminatory. Greater exposure was also increased the likelihood to externalize feelings and express anger or internalize feelings (Brondolo et al., 2005). Therefore, many African-Americans report a myriad of situations in their daily lives as discriminatory. Despite the chronic stressors such as racial and ethnic discrimination and low socioeconomic status, African-Americans and Caucasians have similar rates of psychiatric disorders (Lincoln, Chatters, & Taylor, 2003; Keesler, Mikelson, & Williams, 1999; Keesler, et al., 2003). Epidemiological studies suggest that African-Americans are more likely to endorse somatic rather than affective symptoms (Iwata, Turner, & Lloyd, 2002). In this regard, it is important to examine predictors of psychological distress and coping styles that may differ by ethnicity.

Furthermore, the cardiovascular literature suggests that African-Americans also tend to adopt the active coping strategy of John Henryism in which hard work and determination are used to cope with adversity (e.g., Dressler, Bindon, Neggers, 1998;

Williams & Lawler, 2001). Maladaptive coping strategies are associated with increased health risk (Clark et al., 1999). For example, passive coping strategies (e.g., acquiescence or acceptance of substandard treatment) in response to ethnic discrimination are associated with a four-fold risk of hypertension in African-American women compared to those who used an active coping style (Krieger, 1990). With regard to social support, Lincoln and colleagues (2003) found that positive effects of social support were blunted in circumstances of financial strain and traumatic events for Caucasian Americans. For African-Americans, however, only traumatic events diminished the positive effects of social support. This literature suggests, therefore, that the two ethnic groups not only use different strategies to cope with stress, but also that a similar strategy may yield different outcomes, depending on the ethnicity of the individual.

***Conceptual Model.*** Figure 1 graphically depicts the accepted model of obesity as an interaction among biological, behavioral, and environmental factors. The central research question of this master's thesis is whether the accepted model can be used to partially understand why African-Americans are disproportionately affected by obesity. More specifically, the proposed model as shown in Figure 2 superimposes the factor of Ethnicity onto the accepted model. Whether ethnicity (African-American or Caucasian) strengthens the relationship among known factors associated with the development of obesity is unknown.

*Biological factors.* Obesity affects how energy is regulated in response to normal cycles of rest, eating, and physical activity. The research literature suggests that obesity is associated with decreased insulin sensitivity and poor control of basic activity of the HPA axis. Therefore, energy-regulating HPA axis hormones and insulin were examined

in this study. Evidence suggests that these metabolic processes differ among African-Americans and Caucasians, such that African-Americans have higher levels of fasting insulin and acute insulin responses to a meal and blunted cortisol responses to HPA axis stimulation. Chronic HPA axis activation may further decrease insulin sensitivity in African-Americans. Increased insulin resistance is a marker of the metabolic dysregulation associated with obesity.

*Environmental factors.* External events or stimuli that are appraised as stressful and exceeding an individual's coping resources may affect body weight are the focus of this study. Researchers tend to agree that African-Americans report higher levels of chronic stress compared to Caucasians. These differences have been attributed to additional sources of chronic stress such as racial discrimination and low socioeconomic status that are more likely to be experienced among African-Americans. Metabolic processes associated with chronic stress may promote the development of obesity.

*Behavioral factors.* If it is true that chronic stress promotes obesity, then it is also important to determine whether coping styles differ by ethnicity. Adaptive coping styles and healthy behaviors such as seeking social support, and proper nutrition and exercise can be protective against the effects of chronic stress. Therefore, the Stress Profile was used to determine levels of chronic stress and coping behaviors/styles. It also follows that if African-Americans report higher levels of perceived chronic stress, then African-Americans also will have less effective coping strategies than will Caucasians.

***Summary of Conceptual Model.*** Taken together, the proposed conceptual model suggests that ethnicity strengthens the relationship among the biological, environmental, and behavioral correlates of obesity. It is predicted that African-Americans will have

higher insulin responses to a meal, a greater number of environmental events appraised as stressful, and fewer resources to manage events or stimuli that have been appraised as stressful compared to Caucasians. The implication is that the cumulative effect of these biological, environmental, and behavioral factors is a psychological and metabolic profile that is more obesity-promoting among African-Americans than Caucasians.

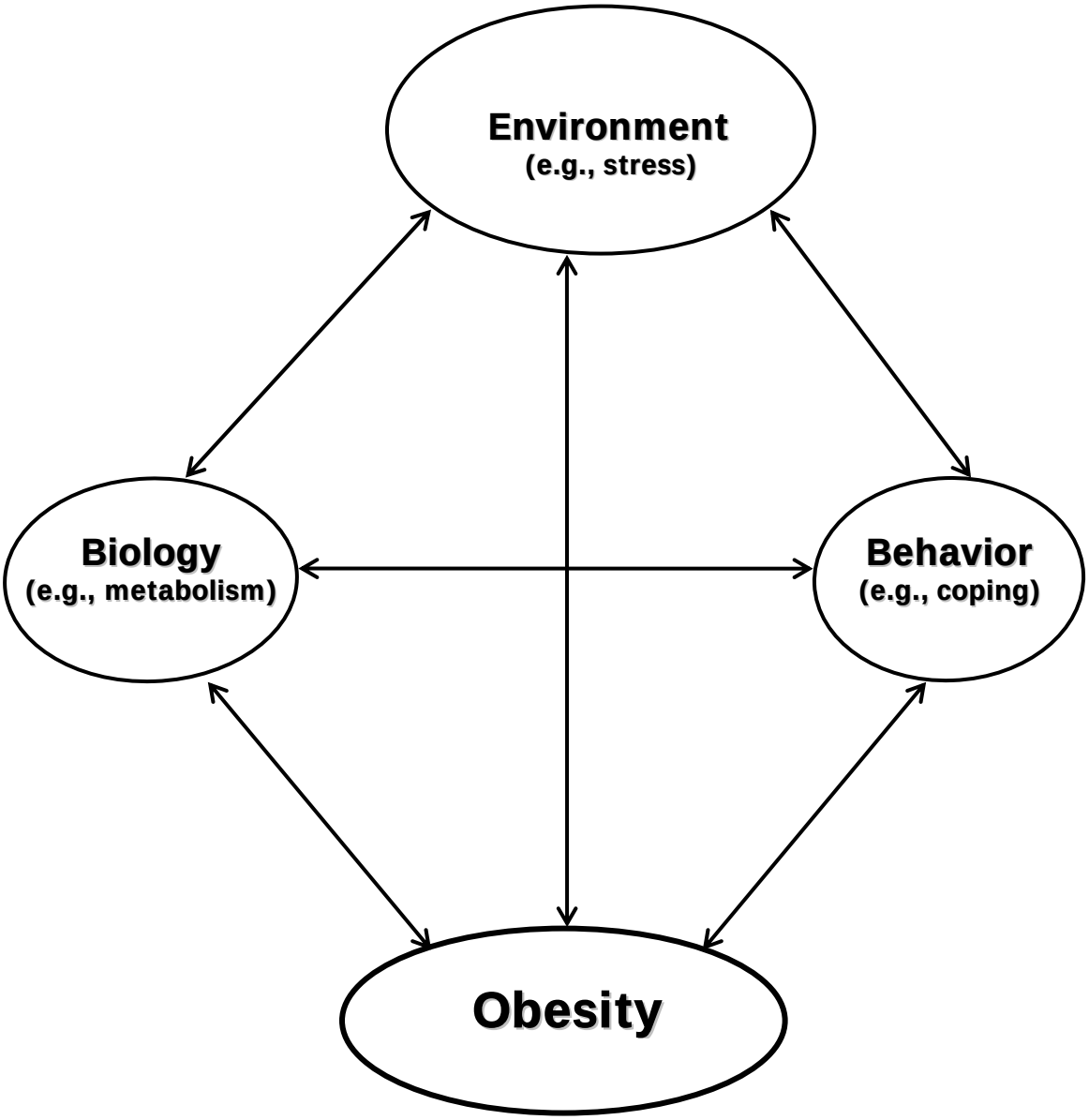


*Figure 1.* Model depicts causes of obesity as an interaction among biological, behavioral, and environmental factors.

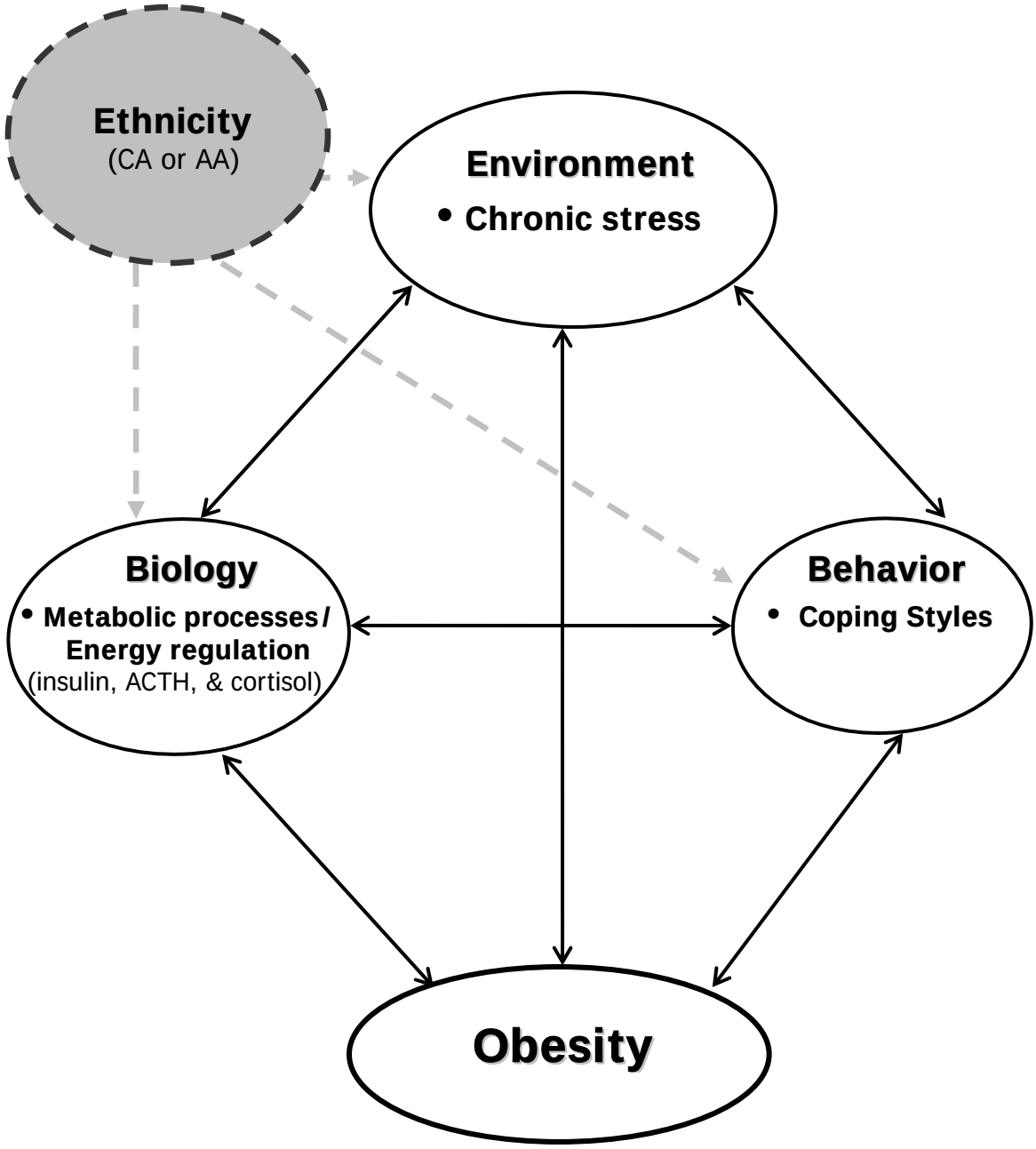


*Figure 2.* Conceptual Model of Master's Thesis.

**Rationale for Procedures**

To determine whether the biological factors associated with the development of obesity differ among African-Americans and Caucasians, this project used two different stimuli activate metabolic responses: a meal and exercise. These stimuli were chosen because they are metabolically relevant; that is, a meal constitutes a metabolic load and exercise constitutes a metabolic demand. These methods were chosen because they have been widely-used in the literature and reliably activate the HPA axis, as well as other metabolic processes (e.g., Deuster et al., 1989, 1992, 1998, 2000; French et al., 1990; Goetz, French, Thomas, Gingerich, & Clements, 1995).

*Stimulating Metabolic Responses*

**Use of a meal.** It is widely accepted that feeding stimulates the HPA axis (Korbonits et al., 1996). Many studies have used a meal stimulus to examine HPA axis reactivity. Typically, a carbohydrate-rich, protein-rich, or mixed (e.g., contains all three macronutrients: carbohydrates, protein, and fat) meal is used (e.g., Korbonits et al., 1996; Pasquali et al., 1998). The meal consists of about 700-800 kilocalories and is consumed within a 5-15 time period. Most studies require that participants fast at least 12 hours prior to consuming the standardized meal.

*Differences between normal weight and obese individuals*. Normal weight and obese individuals exhibit different metabolic responses to a meal. Korbonits and colleagues (1996) found hypocortisolemia in obese individuals at baseline, but significantly greater cortisol levels after the meal and longer recovery times to pre-meal baseline rates for obese compared to lean subjects. In another study, obese men had higher ACTH and no difference in cortisol levels compared to normal controls (Pasquali

et al., 1999). Other researchers have found that the HPA response to feeding differs according to body fat distribution and the macronutrient composition of the meal. In a study that used a mixed meal (i.e., 40 g of protein, 31 g of lipids, and 90 g of carbohydrates), premenopausal women with abdominal obesity had higher ACTH pulsatile secretions in the morning, but ACTH and cortisol levels were similar to obese women with a subcutaneous body fat distribution (Pasquali et al., 1998). These investigators also reported that women with abdominal obesity had a more rapid rate of increase in cortisol after lunch than after dinner. Others have reported that only a high-carbohydrate meal, not a meal high in lipids and proteins, significantly activated the HPA axis in individuals with abdominal obesity but not in those with peripheral obesity or normal controls (Vicennati et al., 2002). Levels of ACTH, cortisol, insulin, or glucose did not differ between obese individuals with abdominal body fat distribution and those with peripheral body fat distribution before the meal challenge (Vicennati et al., 2002).

*Ethnic differences*. To date, no laboratory studies have examined metabolic responses to a meal challenge in overweight or obese non-diabetic African-American adults**.** The extant literature in the area of insulin responses of African-Americans focuses on normal weight or co-morbid obese and diabetic subgroups. These studies also rely on correlational data and insulin or glucose infusions to measure related responses. However, with the growing concern of childhood obesity and type II diabetes, some studies have examined insulin responses in African-American children. In a study of obese African-American and Caucasian children, obese Caucasian children demonstrated less insulin resistance (i.e., insulin was relatively efficient at uptake of glucose) in response to a high-dextrose drink (Preeyasombat et al., 2004).

**Use of exercise.** Since the 1970s, exercise has been considered a reliable and effective activator of the HPA axis (Brandenburger & Follenius, 1975; Wittert, Livesey, Epiner, & Donald., 1996; Brown, Tyler, & Deuster, unpublished manuscript). Numerous studies use an exercise challenge to stimulate the HPA axis. Cortisol levels increase in response to moderate to high intensity exercise (e.g., greater than 50% of $VO_2$max) but decline in response to low intensity exercise (e.g., less than 50% of $VO_2$max) (McMurray & Hackney, 2005). Given the moderate to high intensity exercise conditions in the present study, it was expected that cortisol levels would increase across all groups. Participants usually are subjected to a standardized exercise test in which a treadmill or stationary bicycle (cycle ergometer) is used to control and to monitor the participant's workload. There are two types of standardized exercise tests: maximal or submaximal. The maximal exercise test progressively increases the speed and workload until exhaustion to determine the participant's maximal aerobic capacity ($VO_2$max). A device that measures the volume of oxygen and carbon dioxide expired is attached at the mouth and yields the participant's $VO_2$max. Exercise intensities are calculated based on the individual's physical fitness level. Commonly used exercise intensities for the submaximal exercise test are 50%, 70%, and 90% of $VO_2$max (Brown, Tyler, & Deuster, unpublished manuscript). At exercise intensities of at least 70%, various metabolic responses such as an increase in ACTH and cortisol have been consistently documented in the literature (Brown, Tyler, & Deuster, unpublished manuscript; Giannopoulou, Carhart, Sauro, & Kanaley, 2003).

*Ethnic differences.* Few studies have assessed effects of exercise in Caucasians compared to African-Americans. One such study examined responses of women.

Similar to the previously mentioned studies that stimulated the HPA axis using synthetic hormones, exercise induced greater concentrations of ACTH in African-American women than in Caucasian women without any significant differences between the groups in cortisol levels at pre- or post-exercise conditions (Yanovski et al., 2000). However, a limitation of this study is that cortisol was only measured immediately and 10 minutes after exercise. It may be that differences in cortisol responses exist between these two ethnic groups at later time points not measured in this study.

In a study of African-American and Caucasian adolescents, participants completed a maximal treadmill test. In response to this metabolic demand, African-American girls had the highest insulin concentrations and Caucasian girls had the lowest (Gutin et al., 2004). However, this relationship disappeared when fitness level and body fat percentage were covaried. Fasting insulin level also was positively correlated with body fat percentage, independent of gender or ethnicity. These findings are consistent with the epidemiologic data on the prevalence rates of diabetes and obesity in adults.

This research project assessed: (1) metabolic responses to energy intake and energy expenditure of African-American men and women compared to Caucasian-American men and women in individuals with BMIs ranging from normal to obese; and (2) the relationship of environmental variables, such as social support and health habits, to the metabolic responses and to BMI in African-American men and women compared to Caucasian men and women in individuals with BMIs ranging from normal to obese. Metabolic responses assessed included adrenocorticotropin hormone (ACTH), cortisol, and insulin. These indices were assessed basally, in response to a meal, and in response to exercise. The primary aim of this study was to examine the psychological and

metabolic factors that might contribute to the prevalence of overweight and obese African-Americans.

*Hypotheses*

The central hypothesis of this project is that hormonal dysregulation, precipitated or exacerbated by chronic stress, may contribute to conditions of overweight and obesity (conditions that are prevalent in the African-American population). This project focuses, in part, on the HPA axis because it plays central roles in the regulation of metabolism, energy expenditure and fat deposition as well as in responses to chronic stress. Further, HPA axis abnormalities have been implicated in obesity, insulin resistance, and psychological disorders.

Therefore, the first purpose of this project was to assess chronic stress levels in Caucasian and African-American men and women. The second purpose of this Master's project was to characterize and compare hormonal and metabolic responses of Caucasian and African-American men and women to two metabolically-relevant events — a metabolic load (standardized meal) and a metabolic demand (standardized exercise). The hormonal and metabolic responses included HPA axis hormones (i.e., ACTH and cortisol) and insulin responses to a meal. The third purpose of this project was to assess the relationship among BMI, chronic stress, and metabolic responses.

**Hypothesis 1:** African-Americans will report higher levels of perceived chronic stress than will Caucasians.

**Hypothesis 2:** African-Americans will have less effective coping skills than Caucasians.

African-Americans reported greater levels of chronic stress, fewer hours of rest and sleep, less social support, and more negative appraisal of life events than did Caucasians.

**Hypothesis 3:** African-Americans will have metabolic profiles that are more obesity-promoting than will Caucasians.

**- 3a:** African-Americans will have greater insulin responses to a standardized meal than will Caucasians.

**- 3b:** African-Americans will have greater ACTH responses to exercise and a meal than will Caucasians, but the two groups will have similar cortisol responses to exercise.

**Hypothesis 4:** Chronic stress levels will be related to BMI and to metabolic responses.

- **4a:** Chronic stress levels will have a significant positive correlation with BMI in African-Americans and Caucasians.

- **4b:** Chronic stress levels will have a significant positive correlation with cortisol in African-Americans and Caucasians.

- **4c:** BMI and cortisol will have a significant positive correlation in African-Americans and Caucasians.

# METHODS

*Design and Sample Size*

The study was a 2 x 2 x 2 mixed factorial design with a between-subjects factor of Ethnicity (African-American or Caucasian) and within-subject factors of time (Visit 1 or Visit 2) and type of metabolic challenge (standardized liquid meal or exercise). There were 31 African-American participants (15 men; 16 women) and 32 Caucasian participants (19 men; 13 women). These data were collected as part of a larger study involving two additional laboratory visits that assessed the effects of glucocorticoid receptor blockade on these responses. In the larger study, therefore, data were collected from participants during four laboratory visits – a baseline visit and three additional visits under three experimental conditions (placebo and two different glucocorticoid receptor blockers administered in counterbalanced order). This master's project consists of data collected during the baseline and placebo condition visits. Because the timing of the placebo visit differed across participants, ranging from visit 2 to visit 4, responses also were analyzed to test for effects of visit as a separate independent variable. With regard to this master's project, all participants were tested on separate days under two different conditions: maximal exercise test and submaximal exercise test. The maximal exercise test was used to determine the workload for the subsequent submaximal exercise test. For the remainder of this paper, exercise challenge is used in reference to exercise as a physical stimulus that activates the HPA axis.

Sample size was determined based on three criteria: (1) alpha level of 0.05 (two-tailed tests) and power of 0.80 according to standard statistical procedures; (2) review of the relevant literatures on ethnic differences in obesity, chronic stress, and hormonal

responses (e.g., insulin, ACTH, cortisol); (3) and review of data from previous endocrine and metabolic studies in the Uniformed Services University of the Health Sciences (USUHS) Human Performance Lab. Estimates of effect size were determined by calculating an estimated omega squared. Phi statistics then were calculated to determine the ratio of treatment variance to error variance for a given sample size. Using phi and power tables, the necessary sample size to achieve power of 0.80 was determined. Based on the literature and these calculations, a minimum of 12 subjects per cell were necessary to detect ethnic differences in hormonal responses. The Institutional Review Board of USUHS approved this sampling plan as well as all study procedures and methods.

*Participants*

African-American and Caucasian participants between the ages of 18 and 45 years old were recruited via local newspapers, from local universities, churches, and the surrounding metropolitan area. All participants determined their ethnicity by self-report. A total of 262 participants were screened over the telephone and those who met the inclusion criteria and who were willing to participate gave written informed consent. This master's project only includes a subset of participants (63 of 78) who had complete psychological and metabolic data for the initial and placebo visits.

*Measures*

**Stress Profile.** The Stress Profile was used to quantify chronic, perceived stress across numerous domains. Each participant completed the Stress Profile once on the first visit prior to exercise testing. The Stress Profile has 123 items that provide scores in 15 areas related to perceived stress and health risk, with measures for response bias and inconsistency (Nowack, 1999). It also is a reliable and valid indicator of stress

vulnerability and resistance. It was standardized on a nonclinical adult sample of various ethnic groups. This self-report questionnaire includes an inconsistent responding index, which consists of 10 items that have similar content and theoretically should be answered similarly. There also is a response bias index, which contains five true-false items and determines whether respondents are trying to present themselves in an unrealistically favorable manner. Internal consistency is satisfactory (r = 0.72), with a range from r = 0.51 to 0.91. The Stress Profile has adequate reliability (r = 0.76 - 0.86) and has been validated on an ethnically-diverse population. There were no significant gender or ethnic differences in the standardization sample (Nowack, 1999). The content of each subscale is described below.

*Stress.* There are six items that reflect perceived stress experienced in the last 3 months. Health, work, financial, family, social, and environmental hassles are assessed for frequency using a 5-point Likert scale from never to always. None of these items directly ask about major life changes. Therefore, it is possible that individuals with low scores have major life events occurring in their lives that are not perceived as highly stressful. This is an important distinction considering that health status is associated with perceived levels of stress.

*Health habits.* This subscale contains four primary areas (e.g., exercise, rest/sleep, eating/nutrition, prevention) that yield individual scores for specific health-promoting behaviors.

*Exercise.* Three items assess whether respondents engage in at least 20-30 minutes of leisure physical activity, cardiovascular exercise, and muscle tone/strength training at least two or three times per week.

*Rest/sleep.* Five items address the amount and quality of sleep and relaxation.

*Eating/nutrition.* Scores on the eating/nutrition subscale indicate dietary practices. Five items assess whether meals are well-balanced (i.e., intake of saturated fats, caffeine, sugar, salt, and total calories) and eaten regularly.

*Prevention.* There are 11 items that assess preventive health and hygiene, such as medication compliance and routine medical checkups to reduce the risk of disease. Other items yield information about the use of cigarettes, recreational drugs, and alcohol in a 24-hour period.

*Social support network.* This subscale contains 15 items that assess the emotional support the respondent receives from his/her immediate environment.

*Type A behavior.* In this subscale, characteristics of Type A behavior such as cynical mistrust, anger, and hostility are assessed. The ten items measure internalized anger, expressed anger, time urgency, and competitive behaviors. A sample item is "My activities and schedule push me to be as busy and active as possible, stretching me to the limits of my energy and capacity."

*Cognitive hardiness.* The cognitive hardiness subscale has 30 items about attributions, attitudes, and beliefs. High scores reflect individuals who view themselves as having a sense of control over major life events and who perceive challenges as opportunities to succeed. Low scores reflect individuals who are vulnerable to feeling alienated in their social roles at work and home.

*Coping style.* The coping style subscale consists of four subscales (e.g., positive appraisal, negative appraisal, threat minimization, and problem focus) that address specific coping strategies.

*Positive appraisal.* Positive appraisal is characterized by employing supportive and encouraging self-talk to cope with a challenging situation. Individuals who employ positive appraisal are able to generate favorable outcomes. There are five items on this subscale.

*Negative appraisal.* Five items assess the tendency to approach a difficult life situation with self-blame, criticism, or catastrophic thinking. This coping strategy focuses on worst possible outcomes.

*Threat minimization.* Threat minimization also is referred to as avoidance. This coping strategy emphasizes the humor in problematic situations and often distracts attention from the stressor. There are five items on this subscale.

*Problem focus.* The four items assess the degree to which an individual devises a specific course of action to reduce the impact of current problems. This coping style emphasizes the tendency to try improving the troublesome situation.

*Psychological well-being.* There are 12 items on the psychological well-being subscale. It is an overall assessment of life satisfaction in the past 3 months.

***Profile of Moods State-Short Form (POMS-SF).*** The Profile of Mood States (POMS; McNair, Lorr & Droppleman, 1971) is frequently used in clinical research settings to assess short-term changes in psychological and emotional states. The POMS-SF (Shachman, 1983) is a shortened version, which contains 37 adjectives indicative of six mood dimensions: tension-anxiety, depression-dejection, anger-hostility, fatigue-inertia, vigor-activity, and confusion-bewilderment. A global distress score, known as the total mood disturbance score, is calculated by subtracting the vigor score from the summed total of tension, depression, anger, fatigue, and confusion scales. It uses a 5-

point scale from not at all to extremely to evaluate the respondent's persistent mood and reaction to current life situations. The correlations between the six subscales and the total mood score on the POMS-SF were above 0.95 in five different clinical samples and one sample of healthy controls (Curran, Andrykowski, & Studts, 1995; Shacham, 1983). Although the longer version was standardized on clinical and non-clinical adult samples, there is minimal normative data available for the POMS-SF (Curran, Andrykowski, & Studts, 1995). The available psychometric properties have been provided. However, the POMS-SF is sensitive to acute treatment effects. Therefore, it was administered upon arrival to the laboratory, after exercise testing, and after the metabolic load demand for visits 1 and 2.

***Beck Depression Inventory (BDI-I).*** The BDI was originally developed in 1961 (Beck, Ward, Mendelson, Mock, & Erbaugh, 1961a). It has been revised to reflect changes in the diagnostic criteria for depression and is widely used to assess severity of depressive symptoms. The standardization sample included African-American and Caucasian men and women (aged 15-44 years) who were psychiatric inpatients and outpatients. Caucasians and low socioeconomic status groups were overrepresented in the original normative sample (Beck, Ward, Mendelson, Mock, & Erbaugh, 1961b), but the original and revised versions have a high correlation ($r = 0.94$) (Lightfoot & Oliver, 1985; as cited in Groth-Marnat, 1990). The revised versions have adequate convergent validity with other depression instruments such as the Hamilton Rating Scale for Depression (range $r = 0.73 – 0.80$) and adequately discriminate self-reported anxiety (Beck, Steer, & Garbin, 1988; Groth-Marnat, 1990). Test-retest reliability ranges from 0.48-0.86, depending on the amount of time between intervals and type of population

(Groth-Marnat, 1990). The split-half reliability coefficient is 0.93. Content validity was obtained by consensus among psychiatrists who conducted a diagnostic interview for depth of depression. There was agreement in 97% of cases. The BDI-I assesses how the participants feel at the present time. Therefore, the BDI-I was administered to control for effects associated with depressed mood. The BDI consists of 21 items and yields scores ranging from 0 to 63. The internal consistency is high for normal populations (r = 0.81) (Kramer & Conoley, 1992). Each participant completed the BDI once per visit prior to exercise testing. Among clinical populations, scores 9 or below are within the normal range. In a non-clinical sample, scores below 15 are within the normal range.

***State-Trait Anxiety Inventory (STAI; Forms Y-1 and Y-2).*** The STAI (Spielberger, 1983) is composed of two separate 20-item scales used to assess individual state and trait anxiety. The normative sample included men and women high school and college students, military recruits, and working adults (aged 19-69 years). The state items measure transient changes in intensity of anxiety with a 4-point scale (e.g., not at all (1), somewhat (2), moderately so (3), and very much so (4) using Form Y-1. The trait items measure the frequency of anxiety using a 4-point scale: almost never (1), sometimes (2), often (3), and almost always (4) using Form Y-2. Both scales have impressive internal consistency from 0.88-0.92 with sample populations (Spielberger & Vagg, 1984). As expected, the test-retest reliability is higher for the trait scale than the state scale. Forms Y-1 and Y-2 were both administered on visit 1. Only Form Y-1 was administered on visit 2. The concurrent validity between the trait anxiety scale and other measures such as the Taylor Manifest Anxiety Scale and IPAT Anxiety Scale is high, ranging from 0.73 – 0.85) (Spielberger, 1983). The test-retest correlations were low for

state anxiety (ranging from r = 0.16 – 0.62) given the transitory construct measured, but the trait-anxiety correlations were higher (ranging from r = 0.65 – 0.86) as expected given the stability of this construct. Other demographic variables about the standardization sample were not provided.

***The Borg Perceived Exertion Scale***. The Borg Rating of Perceived Exertion Scale (RPE; Borg, 1998) was used to document perceived effort and relative exercise intensity. The Borg scores correlate strongly with physiological markers of exercise intensity: 0.62 for heart rate, 0.57 blood lactate, 0.64 for percentage of maximal aerobic capacity, 0.63 for oxygen consumption, 0.61 for ventilation, and 0.72 for respiratory rate (Chen, Fan, & Moe, 2002). At the end of each exercise session, participants were asked to rate the intensity of exercise on a scale from 6 ("no exertion at all") – 20 ("maximal exertion"). A rating of at least 17 is associated with high intensity exercise, which stimulates the HPA axis (ACSM, 2000; Borg, 1982). Under standardized test conditions (e.g., no distractions to interfere with the perception of work), the Borg scale has a high test-retest reliability. For example, the test-retest reliability during cycling tests at progressive workloads with exercise intensities at least 70% maximum heart rate range from r = 0.76 to 0.90 (Scherer & Cassaday, 1999).

*HPA Axis Stimulation*

Two different reproducible and quantifiable laboratory experiences were used to stimulate the HPA axis: a standardized meal constituting a metabolic load and a standardized exercise test constituting a metabolic demand. These manipulations have been widely-used to produce consistent HPA axis activation (Deuster et al., 1989, 1992, 1998, 2000).

***Standardized Meal: A Metabolic Load.*** Participants drank 16 ounces of Ensure-Plus on two occasions: once before the maximal treadmill test and once after the submaximal exercise test. The different orders were used to determine whether the HPA response to exercise differed depending on the presence of a prior metabolic load. Sixteen ounces of Ensure-Plus provides 720 kilocalories comprised of 56.4% carbohydrates, 29% fat, and 14.6% protein. Ensure was available in vanilla, chocolate, strawberry, and butter pecan. This liquid meal has been used previously in other studies and is known to stimulate both glucose and insulin responses (French et al., 2002; Goetz et al., 1995).

***Standardized Exercise: A Metabolic Demand.*** Previous studies indicate that treadmill exercise activates the HPA axis relative to the exercise intensity (Deuster et al., 1989; Negrao, Deuster, Gold, Singh, & Chrousos, 2000; Petrides et al., 1994). Specifically, exercise intensities from 50-90% $VO_2$max stimulate the HPA axis (Negrao et al., 2000). Exercise tests were carried out on a treadmill (Quinton ST-65, Quinton Instrument Company, Seattle, WA). Participants were instrumented with electrodes (Quinton Q-4500) and a portable metabolic unit for continuous monitoring of heart rate and electrocardiogram, and oxygen uptake, carbon dioxide production, and respiratory exchange ratio by open circuit spirometry (KB20-CosMed, Italy).

*Maximal exercise test.* Each participant completed a maximal exercise treadmill test on the first visit. This test involved a 5-minute warm-up (3.0 mph, 2% grade) followed by walking (or running) at 3 to 7 mph (depending on heart rate during the warm-up) at increasing grades (+2.5%) every two minutes until the subject reached volitional exhaustion. Previous studies have confirmed that 90% $VO_2$max exercise

intensity will activate the HPA axis in all participants under placebo conditions (Deuster et al., 1989; Negrao et al., 2000). The maximal exercise test was used to determine 50%, 70%, and 90% workloads ($VO_2$max) to be used in the subsequent submaximal exercise test. A 5-minute cool-down (low intensity - walking at a 2.5 mph, 0% grade) followed the maximal exercise testing. Average $VO_2$max values for healthy individuals range from 35 – 42 mL/kg/min and 28 – 35 mL/kg/min in men and women, respectively (ACSM, 2000). Maximum $VO_2$max criteria were determined by meeting at least three of the five following criteria: (1) plateau of $VO_2$, (2) respiratory exchange ratio greater than or equal to 1.1, (3) age-predicted maximal heart rate (i.e., 220 - age) achieved (4) RPE of 17 or greater (5) lactate value of 8 mmol/L or greater.

*Submaximal exercise test.* On the subsequent visit, participants underwent a 20-minute submaximal treadmill exercise test. In the larger study that examined the effects of glucocorticoid receptor blockade, participants took four placebo pills on the day before and two placebo pills the day of the submaximal exercise test. The exercise involved a 5-minute warm-up at 50% (5% grade), followed by 10 minutes at 70% (10% grade), and five minutes at 90% (10% grade) of their previously determined $VO_2$max, and a 5-minute cool-down (low intensity - walking at a 2.5 mph, 0% grade).

*Biochemical Analysis.* Hematocrit and hemoglobin were measured in blood samples collected from an indwelling catheter in the antecubital vein prior to exercise to ensure that no subject was anemic. None of the participants included in this master's project were anemic; therefore, these data are not presented. Blood for hormones was collected in EDTA, immediately placed on ice and then centrifuged at 3,000 rpm for 15 minutes. All samples were centrifuged within 30 minutes from collection. Glucose was

measured using a 2700 YSI Lactate/Glucose analyzer. Adrenocorticotropin hormone (ACTH), cortisol, and insulin were measured by radioimmunoassay (RIA) (cortisol: MP Biomedicals, LLC, Orangeburg, NY; insulin: Diagnostic Systems Laboratories Incorporated, Webster, TX; ACTH: Nichols Institute, San Juan Capistrano, CA). All plasma was stored at -70°C and assayed in batch to minimize interassay variability. Detection limits for cortisol, ACTH, and insulin were 0.17 μg/dL, 0.22 pmol/L, and 1.3 μIU/mL respectively. All samples were assayed in duplicate.

*Anthropometric measurements.* Height was taken once on the first visit using by standard techniques. All participants were weighed upon arrival at each visit. Bioelectrical impedance analysis (BIA) (RJL Systems, Incorporated; Detroit, Michigan) was used to assess the amount of body fat. To measure body fat percentage, electrodes were placed on the right hand and right foot while the participant was in the supine position and a small electric current was sent through the water contained in muscle and adipose tissue. BIA measures the amount of resistance to the electric current; muscle mass is less resistant than fat. Waist and hip circumferences were measured by using a tape measure and the waist-to-hip ratio was calculated. The smallest portion of the waist and the widest protrusion of the hips/buttocks were measured twice and the averages were recorded. BIA and waist and hip circumference measurements were taken on the second visit only. For this master's project, only BMI values are reported.

***Procedures***

***Visit 1 (Maximal Exercise Day).*** See Table 1. Upon arrival, participants' weight and height were measured and recorded. An indwelling catheter was placed in the antecubital vein of a forearm; the catheter line was kept patent with a 1% heparin lock.

Fasting blood glucose, hematocrit, and hemoglobin levels were obtained from the initial blood draw to ensure that participants were healthy enough to exercise. Then, participants drank 5 mL/kg of their body weight in water to ensure adequate hydration before exercise. Approximately 40 minutes were allotted to allow absorption of the water and stabilization of stress hormones to the insertion of the intravenous catheter.

**Table 1: Visit 1 Timeline**

| Time (min) | Activity |
|---|---|
| -40 | Insert Venous Catheter |
| | POMS-SF |
| -10 | Blood draw (2 mL) |
| -5 | Drink 720 kcal Ensure Plus |
| 0 | Finish drinking Ensure Plus |
| Post Ensure +10 | Blood draw (7 mL) |
| Post Ensure +20 | Stress Profile |
| | BDI-I |
| | STAI |
| Post Ensure +30 | Blood draw (7 mL) |
| Post Ensure +50 | Blood draw (7 mL) |
| | POMS-SF |
| | Hydrate 0.5% body wt |
| Post Ensure +70 | Blood draw (7 mL) |
| Post Ensure +80 | Physical exam |
| | Resting blood pressure |
| Post Ensure +100 | Review max test procedures |
| Post Ensure +110 | Warm-up on treadmill |
| Post Ensure +115 | Start max test |
| Post exercise | Blood draw (4 mL) |
| | Rating of Perceived Exertion (RPE) |
| | Cool down |
| Post-exercise + 5 | Blood pressure |
| | POMS-SF |

During this period, the following psychological questionnaires were administered: Stress Profile, BDI-I, and POMS-SF. Participants then drank the Ensure Plus. All participants ingested the Ensure within a 5-minute period (time=0). The POMS-SF was administered for the second time 50 minutes after the Ensure was ingested. Next, a physician obtained information about the participants' medical history and performed a physical examination. Once cleared for exercise, participants were briefed on the speed and grade changes as they would occur during the maximal exercise test using a standardized script. In this pre-exercise briefing, participants were instructed that

the test would be terminated when they indicated exhaustion (i.e., by grabbing the bar at the front of the treadmill), and they were encouraged to expend their maximal effort. Participants then were fitted with a face mask and electrodes to monitor respiratory gas exchange and heart activity. Approximately 110 minutes elapsed from the ingestion of Ensure to the start of the maximal exercise test. The maximal exercise test began with a 5-minute warm-up and generally lasted between 12-19 minutes. Once the participant indicated exhaustion, a 5-minute cool-down period ensued. Immediately after the test, participants reported their RPE rating. The third POMS-SF was administered during the cool-down period.

The average time to complete the baseline visit was 3 – 4 hours. During the baseline visit, blood was drawn before ingestion of Ensure and at 10, 30, 50, 70, 95 minutes after ingestion of Ensure (time=0) (see Table 1 for blood draw amounts). No more than 150 ml of blood was taken per week. Laboratory visits were separated by at least 3 days to ensure adequate recovery between exercise tests and to prevent carryover effects between study medications that were used in the larger study. Abstinence from caffeine, alcohol, tobacco, and strenuous exercise was required for at least 12 hours before each test period. Spotters and trained medical personnel were present during the exercise test. The exercise equipment was calibrated with gases of known concentrations of oxygen and carbon dioxide and the room air humidity recorded before each exercise test. Data were recorded every second and averaged over 12-second intervals. All participants reported to the laboratory between 0700 – 0900. Participants were encouraged to complete all testing within two to three months to minimize the effect of changes over time.

| Table 2: Visit 2 Timeline | |
|---|---|
| **Time (min)** | **Activity** |
| -65 | Anthropometric measures |
| -60 | Insert Venous Catheter |
| | Blood draw (2 mL) |
| | Hydrate 0.5% body wt |
| -50 | POMS-SF |
| | BDI-I |
| | STAI |
| -30 | Review exercise procedures |
| -15 | Record blood pressure |
| | POMS-SF |
| - 5 | Blood draw (17 mL) |
| 0 | Begin 5-min warm-up at 50% $VO_2$max at 5% grade |
| +5 | Exercise at 70% $VO_2$max at 10% grade |
| +15 | Exercise at 90% $VO_2$max at 10% grade |
| +20 | RPE |
| | POMS-SF |
| | Blood draw (10 mL) |
| Post exercise + 2 | 5-min Cool down |
| Post exercise + 7 | Resting blood pressure |
| Post exercise + 10 | Blood draw (10 mL) |
| Post exercise + 20 | Blood draw (10 mL) |
| Post exercise + 30 | Blood draw (10 mL) |
| Post exercise + 50 | Blood draw (10 mL) |
| Post exercise + 60 | Ingest 720 cal Ensure |
| Post exercise + 65 | Finish Ensure |
| Post exercise + 75 | Blood draw (13 mL) |
| Post exercise + 95 | Blood draw (13 mL) |
| Post exercise + 115 | Blood draw (13 mL) |
| | POMS-SF |
| Post exercise + 135 | Blood draw (13 mL) |

***Visit 2 (Placebo).*** See Table 2. The placebo visit differed from visit 1 (maximal exercise day) in several ways. First, the placebo visit was a submaximal exercise test without a physical examination. Exercise speed and intensity were standardized using the results of the participants' maximal exercise test during first visit. Second, the test examiner terminated the submaximal test after 25 minutes of exercise. Third, the participants completed the exercise test before drinking the Ensure rather than after drinking the Ensure as in the baseline visit. Participants were on the treadmill 1 hour after arriving at the laboratory. They waited an hour after exercising to drink the Ensure. The average visit lasted from 4-5 hours. Five blood draws were taken post-exercise and four blood draws are taken after the metabolic load at

10-20 minute intervals. The POMS-SF was administered at baseline and after each stressor.

**Data Analyses**

The computer package SPSS was used for all statistical analyses. Chronic stress data were extracted from the Stress Profile. These data were analyzed using analyses of variance (ANOVAs) with between-subjects factor of Ethnicity. A similar approach was used to examine information from other questionnaires (e.g., depression level from the BDI-I; anxiety level from the STAI). Analyses were run on all participants to-date. Participants are still being recruited for the larger study. An alpha level of 0.05 (two-tailed) was used for all statistical tests.

Chi-square analysis was used to assess for differences in ethnic distribution across the timing of the placebo visit (i.e., visit 2, 3, or 4 of the larger study) was similar for African-Americans and Caucasians. Separate repeated-measures ANOVAs were used to analyze ACTH, cortisol, and insulin responses during the maximal (Visit 1) and submaximal (Visit 2) sessions with the between-subjects factor of Ethnicity, and the within-subjects factor of Time. Acute insulin responses were expressed as peak responses and also as areas-under-the-curve (AUCs). The AUCs were calculated by the trapezoidal rule for positive incremental area (Allison, Paultre, Maggio, Mezzitis, & Pi-Sunyer, 1995). These data were analyzed with analyses of variance (ANOVAs) with the between-subjects factor of Ethnicity. Relationships among chronic stress level, psychological and behavioral factors, BMI, and hormonal responses as peaks were examined using Pearson product-moment and Spearman correlations. These analyses were carried out on the subset of participants for whom hormonal data were assessed.

# RESULTS

**Participant Demographics**. Table 3 presents demographics of the participants. Chi-square analysis revealed that there were no ethnic differences in the timing of the placebo visit. There was a total of 63 participants (African-Americans n = 31; Caucasians n = 32). African-Americans tended to be older [$F(1, 61) = 3.138$, $p = 0.08$] than Caucasians. African-Americans also had significantly lower $VO_2$max values [$F(1, 61) = 9.639$, $p < 0.05$], suggesting that overall they were less aerobically fit than were Caucasians. There were no ethnic differences in BMI.

***Beck Depression Inventory (BDI-I).*** See Table 4. The BDI was given once on each testing day. Questionnaires with more than two missing items were considered invalid and excluded from analyses for that particular time point. There were no differences between groups in depression scores on either visit. Group means were compared to the clinically significant population cut-off score of 10 for depression. Both ethnic the groups scored significantly below the cut-off for clinical depression on visit 1 and visit 2.

***State Trait Anxiety Inventory (STAI).*** See Table 5. Mean scores on the state and trait anxiety questionnaires were computed for ethnic and gender subgroups. The mean of each subgroup was compared to the appropriate age-normed and gender-normed sample for healthy, working adults using a one-sample t-test. For one or two omitted items, the mean weighted score was determined. If three or more items were omitted, then these questionnaires were excluded for that particular time point due to the high probability that these questions were answered unreliably. The participants in this study

had lower or similar anxiety levels compared to the normative sample. There were no ethnic differences on state and trait anxiety.

***Stress Profile.*** See Table 6. Scores were obtained on each subscale following procedures outlined in the manual (Nowack, 1999). If scores on the inconsistent responding and response bias indices were 3 or above, then the likelihood that questions were answered unreliably was 68% or more (Nowack, 1999). Four participants scored 3 or greater on at least one of these internal reliability measures. Therefore, the Stress Profile subscale scores for these participants were excluded from data analyses. Stress Profile data were analyzed with multivariate analyses of variance (MANOVAs) with factor of Ethnicity.

The main effect of Ethnicity indicated that African-Americans reported greater levels of chronic stress [$F (1, 61) = 5.181$, $p < 0.05$)], fewer hours of rest and sleep [$F (1, 61) = 10.801$, $p < 0.05$], less social support [$F (1, 61) = 4.613$, $p < 0.05$)], and more negative appraisal of life events [$F (1, 61) = 6.335$, $p < 0.05$)] than did Caucasians.

***Profile of Mood States (POMS-SF)***. See Tables 7 and 8. Raw and standardized scores were examined. There were no statistical differences between these scores. Total mood disturbance can only be computed using the raw scores. Therefore, only raw scores are reported. If more than 10% of the items were left blank, the questionnaire was considered invalid for that particular time point (McNair, Lorr, & Doppleman, 1992). A total of nine participants (visit 1, $n = 4$; visit 2, $n = 5$) were excluded from data analyses because of procedural or experimental error (e.g., three or more omitted items, difficulties with the blood draws that resulted in a failure to administer the POMS-SF at that particular time point).

*Visit 1.* On the maximal exercise testing day (Visit 1), the POMS-SF was administered in the following order: baseline, post-Ensure, and post-exercise. MANOVAs revealed no significant differences in the seven subscales on the POMS-SF between African-Americans and Caucasians. However, the overall F was not significant.

*Visit 2.* On the submaximal testing day (Visit 2), the POMS was administered in the following order: baseline, post-exercise, and post-meal. MANOVAs revealed no significant differences in the seven subscales on the POMS-SF between African-Americans and Caucasians. However, the overall F was not significant.

***Metabolic Responses.*** Physiological responses were assessed for a subset of participants run in the study to-date. Repeated-measures ANOVAs were used to analyze metabolic responses to the meal and exercise challenges with factor of Ethnicity. When sphericity violations occurred, the Greenhouse-Geisser correction was applied. Univariate analyses were used to determine whether the groups differed at baseline on levels of physiological variables or in their peak responses to the meal and exercise challenges.

Adrenocorticotropin hormone (ACTH). See Figures 3 and 4.

*Visit 1.* Four participants had baseline levels of ACTH that were more than two standard deviation units above the mean on visit 1. These participants were considered outliers and excluded from the data analyses. The available data for visit 1 included unequal groups – seven African-Americans (men: n = 2; women: n = 5) and 21 Caucasians (men: n = 13; women n = 8). The groups did not differ in levels of ACTH at baseline. There was a significant main effect of Time, with ACTH levels increasing sharply from time point 70 to the post-exercise time point [$F (5, 130) = 20.102$, $p < 0.05$].

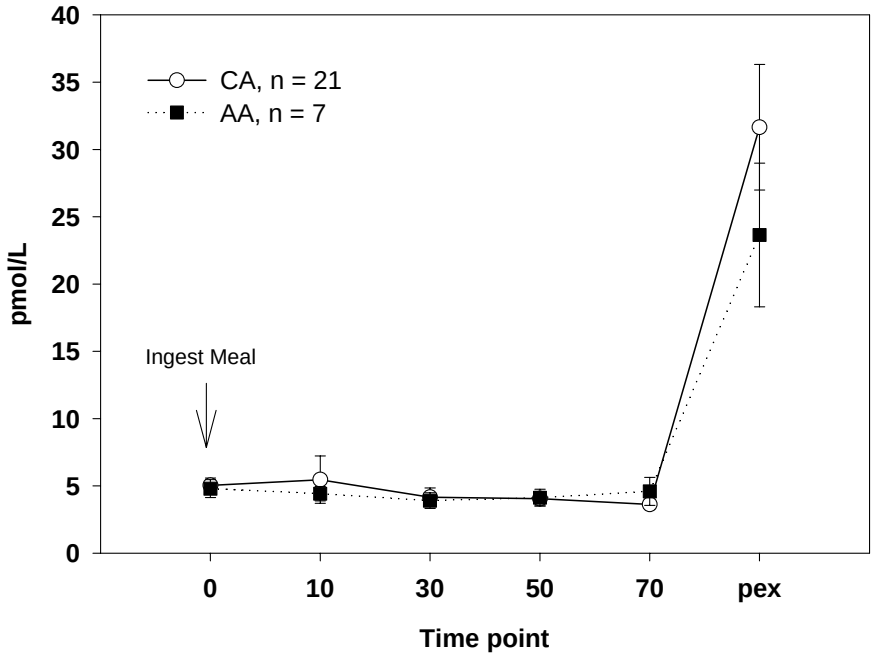


***Figure 3.*** **ACTH Responses on Visit 1. Time is in minutes. Error bars depict standard errors of the mean.**

*Visit 2.* The available data for visit 2 included unequal groups – eleven African-Americans (men: n = 4; women: n = 7) and 28 Caucasians (men: n = 18; women n = 10). The ethnic groups did not differ at baseline in levels of ACTH. There was no main effect of Ethnicity during the course of visit 2. There was a significant main effect of Time, with ACTH levels peaking at post-exercise and then decreasing over the remainder of this testing session [$F (9, 306) = 18.353$, $p < 0.05$]. There was no interaction on visit 2. There were no ethnic differences in peak response to exercise (see time point pex on Figure 4) or a meal (see time point 135 on Figure 4). Although not statistically significant, 70 minutes after the meal African-Americans had a slight increase in ACTH secretion; in contrast, ACTH levels for Caucasians had returned to baseline by this point.

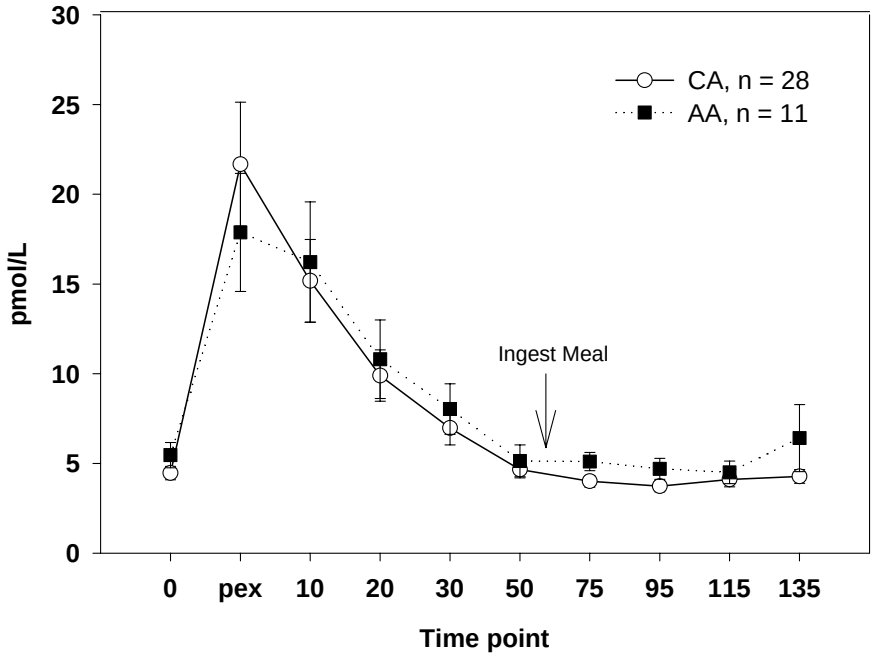


***Figure 4. ACTH Responses on Visit 2.*** **Time is in minutes. Error bars depict standard errors of the mean.**

Cortisol. See Figures 5 and 6. Because of logistical reasons (i.e., the assay kit company could not provide a sufficient number of valid kits), cortisol data were available on visits 1 and 2 for only 25 subjects. The available data included unequal groups – nine African-Americans (men: n = 4; women: n = 5) and 15 Caucasians (men: n = 12; women n = 3).

*Visit 1*. African-Americans had significantly less cortisol at baseline compared to Caucasians [$F(1, 22) = 4.480$, $p < 0.05$]. During this visit, there no significant differences in cortisol responses to exercise or a meal.

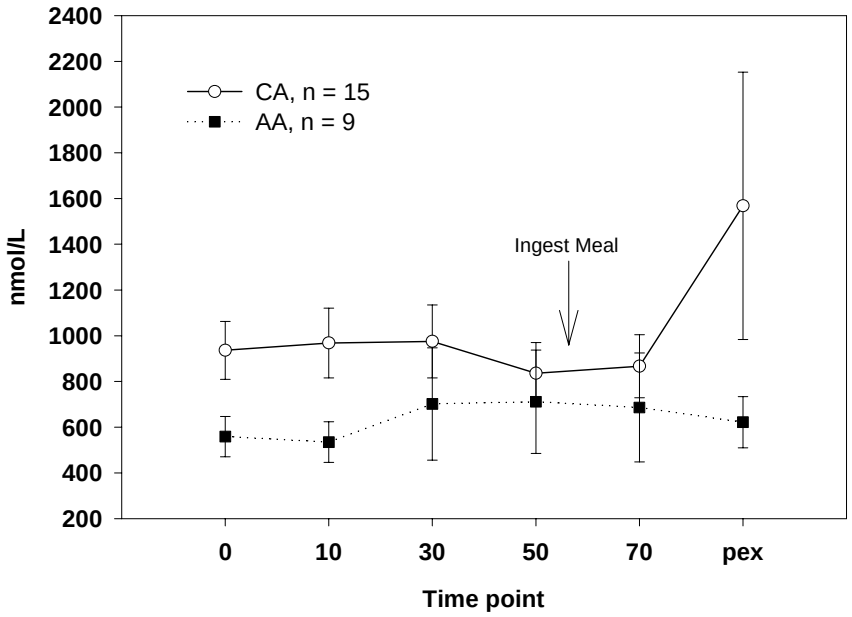


***Figure 5.*** **Cortisol Responses on Visit 1. Time is in minutes. Error bars depict standard errors of the mean.**

*Visit 2.* There were no ethnic differences at baseline or in peak cortisol responses to exercise (see time point 10 on Figure 6) or a meal (see time point 75 on Figure 6).

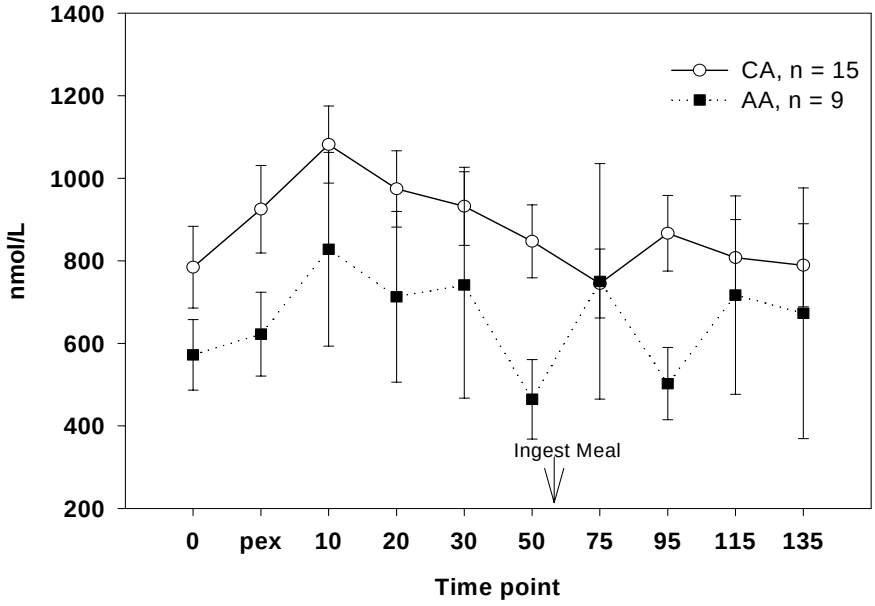


***Figure 6.*** **Cortisol Responses on Visit 2. Time is in minutes. Error bars depict standard error of the mean.**

Acute Insulin Responses. See Figures 7 and 8. The available data for visit 1 included unequal groups – 19 African-Americans (men: n = 10; women: n = 9) and 28 Caucasians (men: n = 18; women n = 10).

*Visit 1.* There was a significant main effect of Time in that insulin levels increased over the initial testing session [F (4, 180) = 43.493 $p < 0.05$]. African-Americans had a higher insulin responses to a meal compared to Caucasians [F (1, 45) = 7.470, $p < 0.05$]. African-Americans exhibited greater peak insulin levels (see time point 30 on Figure 7) than did Caucasians [F (1, 45) = 10.641, $p < 0.05$].

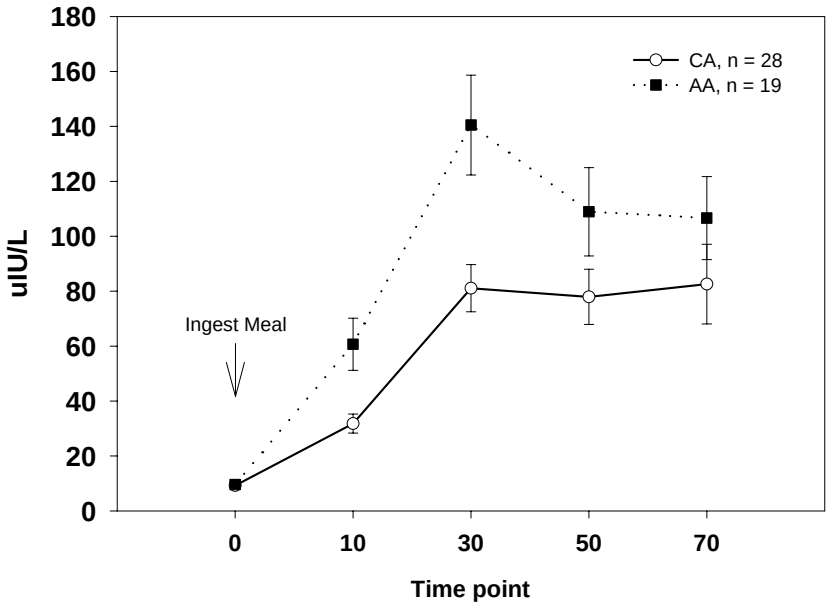


***Figure 7.*** **Acute Insulin Responses on Visit 1. Time is in minutes. Error bars depict standard errors of the mean.**

*Visit 2.* The available data for visit 2 included unequal groups – 20 African-Americans (men: n = 10; women: n = 10) and 29 Caucasians (men: n = 19; women n =10). On visit 2, insulin levels increased over the second visit [F (4, 188) = 38.441, $p < 0.05$]. African-Americans had higher insulin levels after the meal and throughout the

session than did Caucasians [F (4, 188) = 4.480, p < 0.05]. African-Americans had significantly higher peak insulin responses (see time point 30 on Figure 8) than did Caucasians [F (1, 47) = 10.756, p< 0.05].

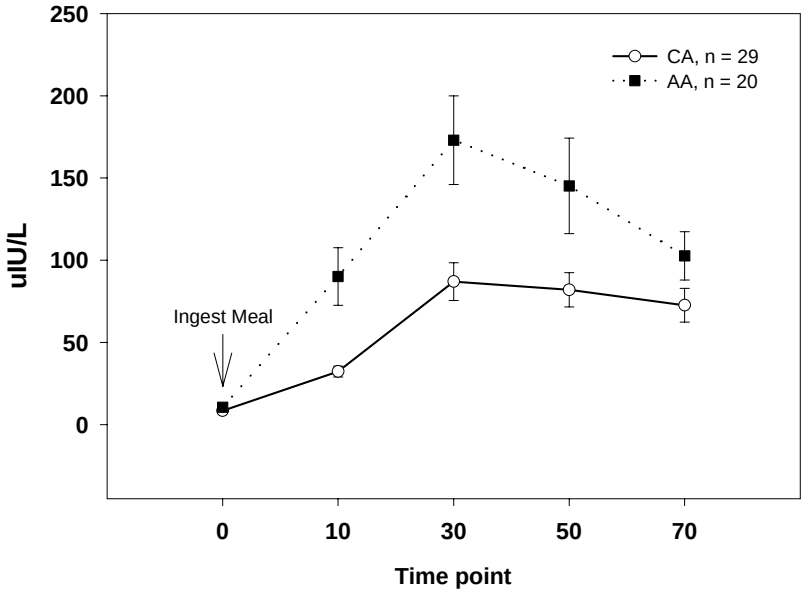


***Figure 8.*** **Acute Insulin Responses on Visit 2. Time is in minutes. Error bars depict standard errors of the mean.**

Total Insulin Production. See Figure 9. African-Americans produced more total insulin than Caucasians [F (1, 45) = 11.599, p < 0.05]. Total insulin production increased from pre- to post-exercise (i.e., visit 1 to visit 2) [F (1, 47) = 3.090, p = 0.052].

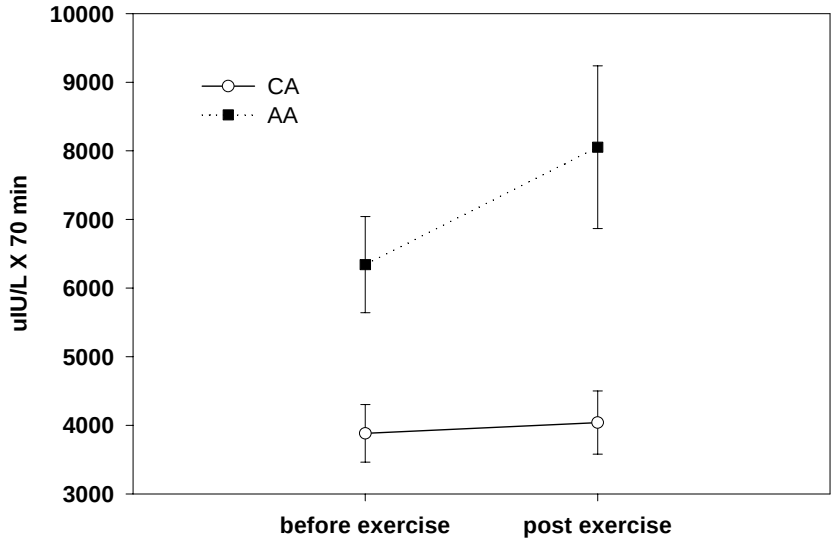


***Figure 9.*** **Total Insulin Production before (visit 1) and after exercise (visit 2). Time elapsed is 70 minutes.**

***Correlations.***

*Stress and BMI.* See Table 9. Pearson's product-moment correlations were performed to assess relationships between the stress subscale on the Stress Profile and BMI by ethnicity. The relationship between stress and BMI was positive but not significant for both African-Americans and Caucasians.

*BMI and basal cortisol.* See Table 9. Spearman's correlations were performed to assess relationships between baseline cortisol levels (i.e., visits 1 and 2) and BMI for each ethnic group. The relationship between BMI and baseline cortisol was negative for both ethnic groups on visit 1. Among African-Americans only, this relationship was significant on visit 2.

*Basal cortisol and Stress subscale.* See Table 9. Spearman's correlations were performed to assess relationships between baseline cortisol levels (i.e., visits 1 and 2) and the stress subscale of the Stress Profile. No correlations were significant.

**Confirmation of Hypotheses**

**Hypothesis 1** that African-Americans would report higher levels of perceived chronic stress than Caucasians was **supported.** The mean score reported by African-Americans on the stress subscale of the Stress Profile was 12% greater than the mean score reported by Caucasians.

**Hypothesis 2** that African-Americans will have less effective coping skills than Caucasians was **supported.** African-Americans reported greater levels of chronic stress, fewer hours of rest and sleep, less social support, and more negative appraisal of life events than did Caucasians.

**Hypothesis 3** that African-Americans would have greater metabolic profiles that were more obesity-promoting than Caucasians was **supported by the insulin responses data**.

**- 3a:** As predicted, African-Americans had higher acute and peak insulin responses to a standardized meal than did Caucasians on both visits. African-Americans also produced greater amounts of total insulin on visits 1 and 2 compared to Caucasians.

**- 3b:** The hypothesis that African-Americans would have greater ACTH responses to exercise and a meal than Caucasians, but that the two groups would have similar cortisol responses was **partially supported**. The ethnic groups did not differ in their ACTH response to a meal or exercise on either visit. As indicated by Figure 4, African-Americans and Caucasians did not differ in cortisol responses to a meal or exercise on visit 1 or 2.

**Hypothesis 4** that chronic stress levels would be related to BMI and to baseline cortisol level was **not supported**. The association between chronic stress level and the primary stress hormone, cortisol, did not emerge as a robust finding.

- **4a:** Chronic stress level was positively correlated with BMI in African-Americans and Caucasians. However, these relationships were not significant.

- **4b:** Chronic stress levels were positively correlated with cortisol among Caucasians on visit 1 only. However, this relationship was not significant.

- **4c:** BMI and cortisol did not have a significant positive correlation in African-Americans or Caucasians. The relationship between BMI and cortisol was significantly negatively correlated among African-Americans on visit 2.

# DISCUSSION

The central hypothesis of this project was that chronic stress may be relevant to many disease states that are prevalent in the African-American population (i.e., obesity) by contributing to metabolic hormonal dysregulation. The project focused, in part, on the HPA axis because it plays central roles in the regulation of metabolism, energy expenditure and fat deposition as well as in responses to chronic stress. This project also focused on insulin responses because as a marker of metabolic dysregulation. More specifically, the purposes of this project were to: (1) assess chronic stress levels in Caucasian and African-American men and women; (2) characterize and compare hormonal and metabolic responses of Caucasian and African-American men and women to two metabolically-relevant events — a metabolic load (standardized meal) and a metabolic demand (standardized exercise); and (3) assess the relationship between BMI, chronic stress and other lifestyle factors, and between metabolic responses, chronic stress, and other lifestyle factors.

The study revealed noteworthy ethnic differences on psychological factors that influence the stress response. African-Americans in this sample reported less social support, less problem-focused coping, fewer hours of rest, and sleep and more negative appraisal than did Caucasians. In addition, African-Americans had higher acute insulin responses and total insulin produced to a meal than did Caucasians. These responses suggest that African Americans may have a greater predisposition toward metabolic disorders such as obesity and insulin resistance. Contrary to the hypothesis, chronic stress was not correlated with level of cortisol. Also, African-Americans had lower

levels of the primary stress hormone cortisol than did Caucasians at baseline and across both visits. Although BMI and chronic stress were positively correlated, the relationship was not statistically significant. Overall, these findings suggest that psychological and biological factors associated with obesity promotion differ between African-Americans and Caucasians. These findings, however, should be interpreted with caution because the study was not sufficiently powered (particularly the hormonal data), and the number of statistical tests may have increased the risk of type I error.

**Psychological data.** *Stress Profile.* Because psychological stress has been implicated in 40% of poor health outcomes (Phillips et al., 2001) and African-Americans reported higher levels of perceived chronic stress than did Caucasians, it is important to consider differences in coping styles. First, African-Americans endorsed having less social support than Caucasians. Data from this study suggest that African-Americans were less likely than Caucasians to benefit from the stress-relieving effects of social support. The influence of social support on stress management and global health has been well-documented. For example, the main effect hypothesis posits that merely having others to confide in and share life experiences with is stress-reducing (Cohen & McKay, 1984). Further, the stress-buffering hypothesis suggests that social support protects against the harmful effects of stress (Cohen & Syme, 1985). Brown & Gary (1988) also found that for African-American women a relatively high number of family members in close geographic proximity, high religiosity, and a strong sense of perceived social support were related to physical health benefits. For African-American men, these factors were not significant. The implication is that African-American men and women have adopted dissimilar coping styles to high levels of perceived chronic stress and that

the coping styles of African-American women are protective, in theory, in terms of developing obesity. Interestingly, however, African-American women have the highest rates of obesity of any subgroup. It is important, therefore, for future studies to directly assess the effectiveness of coping styles in terms of mental and physical health outcomes for African-Americans.

Second, African-Americans in this study reported more negative appraisal compared to Caucasians. Lazarus and Folkman (1984) proposed that cognitive appraisal mediates responses to stress. Situations that threaten physical or psychological well-being can be appraised as either positive or negative. Positive appraisal minimizes the stressor by resulting in an optimistic perspective about the situation; in contrast, negative appraisal is defined by a reliance on catastrophic thinking (Nowack, 1999). Catastrophic thinking adversely affects mood states and is more likely to exacerbate the stress response (Beck, 1995; Lazarus & Folkman; 1984). Therefore, reportedly high levels of perceived stress among African-Americans may have been exacerbated by a tendency to negatively appraise the situation.

Third, African-Americans reported fewer hours of rest and sleep compared to Caucasians. One of the primary benefits of sleep is the reduction of energy intake as well as energy expenditure (Adam, 1998). Longer periods of wakefulness, therefore, might require greater intake of food to sustain energy expenditure. Given the obesity-promoting environment of modern society and high prevalence rates of sedentary lifestyles in America, it is likely that during extended wakefulness calorie-dense foods will be consumed and physical activity will be limited. This combination of factors could be obesity-promoting.

Sleep disturbance also is a marker of poor physical and psychological well-being (APA, 2000; Billiard, 2003; Spiegel, Leproult, & Van Cauter, 1999). Quality sleep serves an important function in restoring cells, tissues, metabolic, homeostatic, and psychic recovery processes in the body (Adam, 1998; Maschke & Hecht, 2004; Rechtschaffen, 1998). Sleep deprivation and sleep debt, in particular, have been linked to metabolic dysregulation (Spiegel et al., 1999). For example, healthy male participants were permitted to accumulate only four hours of sleep per night for six consecutive nights and then were tested for glucose tolerance using a carbohydrate-rich meal. Glucose tolerance decreased by 30% in the sleep-debt condition compared to the sleep-recovery condition (Spiegel et al., 1999). This impairment is similar to levels observed in non-insulin dependent diabetes. In addition, the normal diurnal variations of cortisol were altered by a chronic state of sleep loss such that levels were significantly elevated in the afternoon and evening (Spiegel et al., 1999). High levels of cortisol also constitute part of an obesity-promoting hormonal profile. Apart from other causes, therefore, sleep disturbance and deprivation may place African-Americans at increased risk of developing obesity.

**Biological data.** *HPA axis hormones.* There were no clear ethnic differences in baseline ACTH levels at baseline or in response to a meal or exercise for visit 1 or visit 2. The findings in the present study contradict those reporting higher levels of ACTH between African-Americans and Caucasians, but only one of these studies used treadmill exercise to stimulate the HPA axis (e.g., Yanovski et al., 1995; 1996). Consistent with the literature, the present study also found no differences between ethnic groups in total plasma cortisol concentrations in response to a meal or exercise. However, it is

interesting that African-Americans had significantly lower cortisol levels than Caucasians at baseline despite having similar ACTH levels at baseline. In the present study, cortisol levels did not differ in response to a meal or exercise for either group. However, the high variability in the cortisol data suggests that the sample includes both high and low responders. Several investigators have found that individual responses to HPA axis activation can be divided into subgroups with differential sensitivity (e.g., Kirschbaum et al., 1995; Petrides et al., 1994; Petrides et al., 1997). Therefore, it may be that this sample includes both types of responders.

*Insulin.* It should be noted that the larger study was designed to examine the effects of exercise on insulin sensitivity and whether these effects differed by ethnicity and body type. It was expected that exercise would enhance insulin sensitivity (Mayer-Davis et al., 1998). In order to support this expectation, it was necessary to put procedures in place that would establish how participants' typically respond to a metabolic load and then determine whether this response differed after exercise. Therefore on visit 1, participants ingested the standardized liquid meal prior to exercise. Blood samples were not measured for insulin levels after exercise on the initial visit. The session was terminated shortly after the maximal exercise test. However on the second visit, participants exercised first and then ingested the meal. In this case, the effect of exercise on insulin responses was determined by comparing insulin responses on visit 1 with insulin responses on visit 2.

As expected, both ethnic groups had a substantial increase in insulin secretion in response to a meal independent of visit. Insulin levels had not returned to baseline levels 70 minutes after a meal for either ethnic group. However in response to the meal (on

Visits 1 and 2), African-Americans produced more total insulin and had higher peak insulin responses than did Caucasians. These findings are noteworthy, but the interpretation is complex. Some studies suggest that insulin production declines with a 10-minute bout of submaximal exercise at intensity levels of at least 40% $VO_2$max) (Viru, 1992; Winder, Hickson, Hagberg, Ehsani, & McLane, 1979). The implication is that insulin levels decrease as insulin sensitivity increases in response to exercise (McMurray & Hackney, 2005).

The pattern of increased total insulin production among African-Americans may suggest for this particular group a single bout of moderate to high intensity exercise is either less beneficial for improving insulin sensitivity or requires a longer period to detect the benefits compared to other subgroups. If the former is true, then the long-term consequence may be relatively less efficiency at glucose uptake, indicating a predisposition to dysregulated metabolic states such as diabetes and obesity among African-American, in particular. If the latter is true, then it may be that the present study did not measure responses when they were most likely to occur. For example, enhanced insulin sensitivity – particularly in the skeletal muscle – may (Mikines, Sonne, Farrell, Tronier, & Galbo, 1988) or may not occur (Bonen, Tan, Clune, & Kirby, 1985) immediately after exercise. Furthermore, the gains in insulin sensitivity may persist up to 48 hours post-exercise (Richter, Derave, & Wojtaszewski, 2001). In other words, if exercise-induced insulin sensitivity does have a delayed effect, then the present study would not be able to capture this effect because responses were only measured 70 minutes post-exercise and participants could not retest until at least 72 hours had passed since the initial visit.

*Relationship between psychological and biological data.* Chronic stress and cortisol levels were not related among any groups in this sample. The participants in the present study were matched for body type and had a mean BMI of 27.5 ± 4.6 (range 19.4 – 37.1). Additional subanalyses by body fat distribution once the full study is completed may detect a positive relationship between cortisol and chronic stress levels. According to the Stress Profile manual, scores greater than 60 on the stress subscale are considered high. The average score for this sample was 44.7 ± 10.5 (range 24.0 – 67.0). The implication is that the sample included individuals with relatively low to moderate levels of perceived stress in the past three months. Similarly, this may also explain why chronic stress level and BMI were not correlated. Simply put, participants in this study did not appear chronically stressed.

Although hypercortisolemia commonly occurs in obesity, cortisol levels and BMI were not significantly correlated in the present study. Considering that abdominal fat is associated with greater secretion of cortisol than peripheral obesity (Duclos et al., 2001), body fat distribution may explain why these relationships were not significant. Subanalyses by body fat distribution may reveal a significant positive association between BMI and peak cortisol level in response to a meal and exercise.

*Limitations of the Study*

The limitations of this study should be considered. First, the study was underpowered to detect some of the potential differences between ethnic groups. The present report is based on 63 participants (31 African-Americans and 32 Caucasians), but the biochemical analyses were based on subsets of this group (N = 28 and 39 for ACTH from visits 1 and 2; N = 24 for cortisol; N = 47 and 49 for insulin from visits 1 and 2).

*Post hoc* power analyses revealed that the ACTH and cortisol data were powered between 0.10 and 0.28 with a small effect size. Insulin (for which findings were clear) was powered at 0.72 and 0.82 for the first and second visits, respectively.

Second, the laboratory challenges may limit the generalizability of the study. The type of meal and the exercise condition used as laboratory challenges each are artificial compared with experiences in the real world. As a result, these conditions limit the generalizability of the present study. More specifically, participants were required to drink 720 kilocalories within 5 minutes – not a typical calorie consumption pattern for most people. Typical meals consist of solid and liquid foodstuffs rather than just liquids. Further, obesity-promoting foods are likely to have more powerful hedonic properties than the liquid Ensure-Plus consumed. Including a measure of taste perception would be useful to control for any confounds attributed to the hedonic properties of food. To improve the generalizability of these findings, future studies also could include foods considered more palatable than liquid Ensure. In addition, meals typically are ingested over a much longer period of time than the 5 minutes allotted in this study. Although there was at least an hour between the meal, hydration, and exercise, many participants reported that they "felt full," which may have affected results obtained in this study. The time allowed for calorie consumption, therefore, also should be assessed.

The exercise conditions used in this protocol were tightly controlled, which increased internal validity but perhaps at the expense of external validity. The majority of participants indicated that they participated in regular exercise. However, the exercise tests conducted in this study were at high intensity and most likely exceeded the intensity level of the average exerciser participating in his or her normal routine. Type of exercise

also may be important. Given that exercise is the voluntary movement of muscles in a repetitive and structured way to enhance physical fitness (CDC, 2005), many activities constitute exercise and individuals vary in what activities are considered enjoyable for exercise. Participants in this study were not allowed to choose a type of exercise. It is possible that some participants perceived the treadmill exercise less favorably than other types of exercise.

Third, a measure to capture energy expenditure from routine activities would complement the items on the Stress Profile that assessed participation in different types of exercise. Based on the responses to the self-report items, there were no differences in exercise participation. In this case, it may be that differences in the amount of energy routinely expended on other types of physical activity, such as leisure-time or work-related activities, that were not measured in this study partially explain the findings. Future studies should consider activity monitors, which allow participants free selection of energy-expending activities and quantify energy expenditure associated with daily activities that contribute to energy balance.

Fourth, the relationship between psychological states and obesity-related behaviors such as feeding and activity levels was assessed. We did not, however, manipulate psychological state. Given the comorbidity of mood disorders and obesity, important information could be gained by using psychological state as in independent variable.

Fifth, there are several issues regarding the participants' demographics that should be mentioned. Participants self-identified their race and ethnicity. No additional background information was gathered about the race or ethnicity of parents or

grandparents. Whether these findings are applicable to other ethnic minorities — particular those at risk for metabolic disorders such as Latinos and Native Americans — remains unclear. There may a sampling bias in that many of the participants were recruited via word of mouth at a federal university that caters to training military personnel. In this regard, the results may not be representative of the general population. Participants were not matched based on socioeconomic factors such as education level or income. It may be that these variables moderate the effect of ethnicity on the responses measured.

A number of other variables that could affect results also were not controlled for in this study. Participants were required to abstain from alcohol, nicotine, and caffeine for a minimum of 12 hours prior to exercise testing but abstention from these substances was not verified. Furthermore, it cannot be determined how withdrawal from these addictive substances may have affected participants. Individual diet and nutrition habits outside of the fasting period were not controlled. Also, additional demographic information gathered suggests that at least 16 of the participants were actively trying to lose weight. The exact methods of weight loss and how they may have affected psychological or physiological responses to the study parameters is not known. At least one subject lost 12 pounds between two consecutive visits.

*Future Directions*

The present study was designed to detect ethnic differences in psychological and physiological responses to two laboratory challenges with metabolic relevance. Considering that the difference obesity prevalence rates is most pronounced among African-American women compared to Caucasian women (49% v 31%, respectively;

CDC, 2004), it is important to examine the ethnic-gender interactions. It may that African-American women responded much differently from the other gender-ethnicity subgroups in terms of some psychological (i.e., negative appraisal and social support) and physiological responses to exercise and a meal, but this study was underpowered to detect such a difference.

This study did not directly measure perceived racial and ethnic discrimination or racism-specific coping responses. It is important to quantify these episodes of racism in order to understand their impact on mental and physical health outcomes. The reliance on religious and spiritual coping in the African-American community is well-documented. For example, one study found that African-American churches were more likely to provide mental health services and other support programs than Caucasian churches (Blank, Mahmood, Fox, & Guterbock, 2002). Ellison (1995) found that African-Americans without any affiliation with an organized religion reported higher levels of depressive symptoms than Caucasians. It may be that African-Americans in this study appeared to have less effective coping skills compared to Caucasians simply because this study did not examine religiosity or spirituality.

Furthermore, naturalistic studies that collect stress hormones over a (e.g., 24-hour period) are needed to determine the normal diurnal variation among African-Americans. The present study suggests that African-Americans tend to have blunted morning cortisol levels, which is a pattern similarly observed among obese individuals.

The physiologic profile of the overweight but otherwise healthy African-Americans in this study suggest a high vulnerability toward the early onset of an obesity-promoting metabolic profile. BMI categories were established based on a continuum of

health risks attributed to excess body weight (NIDDK, 2005). Some studies suggested that overweight individuals may not be at a greater risk for certain health outcomes such as mortality compared to normals than previously indicated (e.g., Flegal et al., 2005). However, the findings of the present study suggest that the existing BMI cutoffs may be a liberal indicator of relative health risk for insulin resistance – at least for African-Americans. It may be that for this particular subgroup more conservative cutoffs are necessary to predict associated health risks.

The findings of this study further emphasize the importance of targeted primary and secondary prevention programs for vulnerable populations. Furthermore, the implications are that existing behavioral intervention programs with a sole emphasis on exercise and nutrition may fall short of optimal effectiveness. Ethnic differences in levels levels of perceived chronic stress and responses to cope with chronic stress emerged as a robust finding. In this particular sample of African-Americans, negative appraisal and social support seem to important influences on health behaviors. The implication is that these and potentially other psychological factors may complement a well-managed exercise and nutrition program specifically targeting African-Americans.

Overall, these findings suggest that the proposed model is useful to understand ethnic differences in vulnerability toward the onset of obesity. More specifically, both psychosocial and biological pathways link ethnicity and obesity. Future studies need to triangulate on the complex interactions of these factors. Considering the prevalence of obesity is not expected to level off and ethnic minorities are disproportionately affected (Ogden et al., 2006), the public health imperative is that researchers focus their efforts on

the affected populations using biopsychosocial approaches to understand the correlates associated with the prevention, etiology, and maintenance of obesity.

# Appendix: Tables

| Table 3: Participant Demographics (mean ± standard deviation) | | |
|---|---|---|
| N = 63 | African-Americans | Caucasians |
| Enrolled (n) | 31 | 32 |
| Age (years) | 30.4 ± 7.5 | 27.5 ± 5.3 |
| BMI (kg/m$^2$) | 27.8 ± 4.0 | 26.7 ± 5.1 |
| $VO_2$max L/kg/min | 36.7 ± 9.4 | 43.7 ± 8.3 |
| RPE | 17.4 ± 2.0 | 18.1 ± 1.2 |
| Weight Category | BMI n (range) | |
| Normal weight | 9 (19.9 – 24.7) | 13 (19.4 – 24.1) |
| Overweight | 14 (25.3 – 29.5) | 11 (25.3 – 29.5) |
| Obese | 8 (30.1 – 37.1) | 8 (30.1 – 37.0) |

| Table 4. Beck Depression Inventory (BDI) Scores (mean ± standard deviation) | | | |
|---|---|---|---|
| | F value | African-Americans | Caucasians |
| Visit 1 | F (1,58) = 0.421, ns | 2.4 ± 2.5 | 3.0 ± 3.9 |
| Visit 2 | F (1,56) = 0.004, ns | 2.2 ± 2.9 | 2.3 ± 5.1 |
| Clinically Depressed Cut-off = 10 | | | |
| | African-Americans | Caucasians | |
| Visit 1 | t (30) = - 16.067, p < 0.05 | t (30) = - 10.076, p < 0.05 | |
| Visit 2 | t (31) = - 13.226, p < 0.05 | t (31) = - 8.556, p < 0.05 | |

| Table 5: State Trait Anxiety Inventory (STAI) Scores (mean ± standard deviation) | | | |
|---|---|---|---|
| | F value | African-Americans | Caucasians |
| State (visit 1) | F (1,59) = 0.234, ns | 31.8 ± 10.0 | 30.6 ± 9.1 |
| Trait (visit 1) | F (1,58) = 1.488, ns | 33.5 ± 11.0 | 30.1 ± 10.5 |
| State (visit 2) | F (1, 59) = 0.830, ns | 28.5 ± 7.5 | 26.8 ± 7.5 |

| Table 6: Descriptives for Stress Profile Subscales (mean ± standard deviation) | | |
|---|---|---|
| Subscale | African-Americans | Caucasians |
| Stress | 47.3 ± 11.1 | 41.6 ± 8.8 |
| Health Habits | 55.9 ± 11.3 | 56.1 ± 8.9 |
| Exercise | 53.3 ± 9.3 | 52.0 ± 9.2 |
| Rest and Sleep | 49.4 ± 9.2 | 57.2 ± 9.5 |
| Nutrition | 50.5 ± 9.8 | 51.3 ± 10.2 |
| Prevention | 58.9 ± 15.4 | 60.1 ± 11.6 |
| Addictive Behaviors | 51.0 ± 10.2 | 53.7 ± 8.2 |
| Social Support | 55.4 ± 12.7 | 61.9 ± 11.3 |
| Type A Behavior | 45.4 ± 11.9 | 49.8 ± 10.9 |
| Cognitive Hardiness | 51.9 ± 11.4 | 51.9 ± 11.5 |
| Positive Appraisal | 54.0 ± 9.9 | 50.4 ± 11.4 |
| Negative Appraisal | 44.7 ± 12.5 | 37.2 ± 11.0 |
| Threat Minimization | 51.6 ± 10.1 | 49.1 ± 13.0 |
| Problem-focused Coping | 46.0 ± 10.8 | 49.8± 10.1 |
| Psychological Well-being | 54.1 ± 10.9 | 55.4 ± 12.0 |

| Table 7: Descriptives for POMS-SF Subscale Scores During Maximal Exercise Test on Visit 1 (mean ± standard deviation) | | | |
|---|---|---|---|
| Subscale | Time point | African-Americans | Caucasians |
| Tension – Anxiety | Baseline | 9.9 ± 3.1 | 9.0 ± 2.5 |
| | Post-Ensure | 8.3 ± 2.4 | 8.1 ± 2.5 |
| | Post-Exercise | 8.1 ± 2.6 | 7.6 ± 2.8 |
| Depression – Dejection | Baseline | 10.3 ± 3.0 | 9.1 ± 1.7 |
| | Post-Ensure | 9.2 ± 3.8 | 9.3 ± 2.6 |
| | Post-Exercise | 8.8 ± 1.8 | 9.1 ± 2.3 |
| Anger – Hostility | Baseline | 10.0 ± 3.2 | 9.0 ± 2.0 |
| | Post-Ensure | 8.4 ± 3.1 | 8.4 ± 2.2 |
| | Post-Exercise | 7.7 ± 1.4 | 8.2 ± 2.9 |
| Vigor – Activity | Baseline | 17.6 ± 4.8 | 18.6 ± 5.0 |
| | Post-Ensure | 15.2 ± 5.4 | 19.3 ± 4.9 |
| | Post-Exercise | 15.1 ± 5.1 | 17.0 ± 6.2 |
| Fatigue – Inertia | Baseline | 8.2 ± 2.9 | 7.6 ± 2.5 |
| | Post-Ensure | 7.7 ± 3.4 | 7.2 ± 2.8 |
| | Post-Exercise | 10.9 ± 4.0 | 11.8 ± 4.5 |
| Concentration - Bewilderment | Baseline | 7.2 ± 2.6 | 6.6 ± 2.2 |
| | Post-Ensure | 6.5 ± 3.4 | 6.1 ± 2.0 |
| | Post-Exercise | 5.6 ± 1.5 | 6.4 ± 2.5 |
| Total Mood Disturbance | Baseline | 28.0 ± 11.8 | 22.8 ± 10.8 |
| | Post-Ensure | 24.9 ± 12.0 | 19.8 ± 12.9 |
| | Post-Exercise | 26.0 ± 9.5 | 26.1± 13.7 |

| Table 8: Descriptives for POMS-SF Subscale Scores During Submaximal Exercise Test on Visit 2 (mean ± standard deviation) | | | |
|---|---|---|---|
| Subscale | Time point | African-Americans | Caucasians |
| Tension – Anxiety | Baseline | 7.7 ± 2.8 | 7.3 ± 2.4 |
| | Post- Exercise | 6.8 ± 1.4 | 7.2 ± 2.3 |
| | Post- Ensure | 6.5 ± 0.8 | 6.9 ± 2.7 |
| Depression – Dejection | Baseline | 8.8 ± 2.0 | 8.9 ± 2.4 |
| | Post- Exercise | 8.4 ±1.4 | 9.0 ± 2.0 |
| | Post- Ensure | 8.4 ± 1.5 | 8.8 ± 2.2 |
| Anger – Hostility | Baseline | 8.1 ± 2.1 | 7.9 ± 1.8 |
| | Post- Exercise | 7.6 ± 1.7 | 8.1 ± 2.5 |
| | Post- Ensure | 7.3 ± 1.4 | 7.7 ± 1.9 |
| Vigor – Activity | Baseline | 15.5 ± 4.9 | 19.5 ± 5.4 |
| | Post- Exercise | 15.3 ± 4.9 | 17.5 ± 5.9 |
| | Post- Ensure | 13.2 ± 4.7 | 17.5 ± 6.3 |
| Fatigue – Inertia | Baseline | 8.5 ± 4.6 | 7.3 ± 2.6 |
| | Post- Exercise | 9.5 ± 3.9 | 10.4 ± 4.7 |
| | Post- Ensure | 7.8 ± 4.1 | 7.9 ± 3.2 |
| Concentration - Bewilderment | Baseline | 6.3 ± 2.4 | 5.9 ± 2.2 |
| | Post- Exercise | 5.7 ± 1.8 | 5.9 ± 2.2 |
| | Post- Ensure | 6.0 ± 2.2 | 5.8 ± 1.7 |
| Total Mood Disturbance | Baseline | 24.0 ± 12.6 | 17.9 ± 12.0 |
| | Post- Exercise | 22.8 ± 9.8 | 23.1 ± 14.2 |
| | Post- Ensure | 22.8 ± 10.0 | 16.6 ± 12.4 |

| Table 9: Correlations among Baseline Cortisol, Stress, and BMI | | |
|---|---|---|
| | African-Americans | Caucasians |
| BMI and Stress | + 0.142, ns | + 0.001, ns |
| Stress and Cortisol (visit 1) | - 0.623, p = 0.10 | + 0.056, ns |
| Stress and Cortisol (visit 2) | - 0.467, ns | - 0.214, ns |
| BMI and Cortisol (visit 1) | - 0.633, p = 0.07 | - 0.475, p = 0.07 |
| BMI and Cortisol (visit 2) | - 0.800* | - 0.164, ns |

Significant correlations noted asterisk ($p < 0.05$; ns = not significant); correlations indicate the relationship between the selected factors within each ethnic group, respectively.

## STATEMENT OF WORK

### 1. Year One

a) Ensure that all technical aspects of project are identified and key personnel are aware of expectations and roles by having regular meetings until experiments are underway.
b) Prepare notebook of standard operating procedures.
c) Recruit a postdoctoral fellow.
d) Train all personnel in human use, data management, procedural issues.
e) Order supplies and other biochemical reagents required for initiating the study.
f) Recruit/Screen/Test 10 Overweight/Obese and 10 Non-obese subjects.

### 2. Year Two

a) Recruit/Screen/Test 10 Non-obese subjects.
b) Recruit/Screen/Test 30 Overweight/Obese subjects.
c) Evaluate, reduce, and analyze data for first 20 subjects.
d) Begin biochemical analyses.
e) Begin statistical analyses by body mass index.
f) Examine data on HPA reactivity from the exercise and meal challenge tests as a function of weight status after 30 overweight/obese/non-obese subjects have been tested.
g) Examine data describing HPA axis resistance to feedback control and insulin resistance as a function of weight status after 20 overweight/obese and 15 non-obese subjects have been tested.
h) Examine data describing relation between exercise-associated increases in insulin sensitivity and glucocorticoid sensitivity as a function of weight after 30 overweight/obese and 15 non-obese subjects have been tested.

### 3. Year Three

a) Recruit/Screen/Test 10 Non-obese subjects.
b) Recruit/Screen/Test 30 Overweight/Obese subjects.
c) Continue evaluating, reducing, and analyzing data.
d) Continue with biochemical analyses.
e) Begin statistical analyses on ethnicity.
f) Examine data on HPA reactivity from the exercise and meal challenge tests as a function of ethnicity after 15 AA and 15 CA subjects have been tested.
g) Examine data describing relation between HPA axis resistance to feedback control and insulin resistance as a function of ethnicity after 15 AA and 15 CA subjects have been tested.
h) Examine data describing relation between exercise-associated increases in insulin sensitivity and glucocorticoid sensitivity as a function of ethnicity after 15 AA and 15 CA subjects have been tested.

### 4. Year Four

a) Recruit/Screen/Test 10 Overweight/Obese and 10 Non-obese subjects.
b) Complete subject recruitment.
c) Complete subject testing.
d) Continue evaluating, reducing, and analyzing data.
e) Continue with biochemical analyses.
f) Continue statistical analyses of ethnicity/obesity and potential interactions.
g) Reduce and interpret data on HPA reactivity from the exercise and meal challenge tests as a function of ethnicity and obesity after all subjects have been tested.
h) Reduce and interpret data describing relation between HPA axis resistance to feedback control and insulin resistance as a function of obesity and ethnicity after all subjects have been tested.
i) Reduce and interpret data describing relation between exercise-associated increases in insulin sensitivity and glucocorticoid sensitivity as a function of weight and ethnicity.
j) Examine data as a function of gender after 20 men and 20 women have been tested.
k) Prepare report on results.

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